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Aspiration Pneumonitis and Aspiration Pneumonia—Marik PE. N Engl J Med 2001;344(9): 665–671.

Volume of Air in a Lethal Venous Air Embolism—Toung TJ, Rossberg MI, Hutchins GM. Anesthesiology 2001;94(2):360–361.

Asthma Control: Where Do We Fail? (editorial)—Kips JC, Pauwels RA. Eur Respir J 2000; 16(5):797–798.

Inhaled Nitric Oxide in Adults with the Acute Respiratory Distress Syndrome—Markewitz BA, Michael JR. Respir Med 2000;94(11):1023–1028.

Lung-Protective Ventilation in Acute Respiratory Distress Syndrome: Protection by Reduced Lung Stress or by Therapeutic Hypercapnia? (editorial)—Hickling KG. Am J Respir Crit Care Med 2000;162(6):2021–2022.

Legal Aspects of Withholding and Withdrawing Life Support from Critically Ill Patients in the United States and Providing Palliative Care to Them—Luce JM, Alpers A. Am J Respir Crit Care Med 2000;162(6):2029–2032.

Proceedings of the ATS Workshop on Refractory Asthma: Current Understanding, Recommendations, and Unanswered Questions. American Thoracic Society. Am J Respir Crit Care Med 2000;162(6):2341–2351.

End-Tidal CO_2: Physiology in Pursuit of Clinical Applications (editorial)—Levine RL. Intensive Care Med 2000;26(11):1595–1597.

Lung-Protective Mechanical Ventilation Strategies in ARDS—Lee WL, Detsky AS, Stewart TE. Intensive Care Med 2000;26(8):1151–1155.

Interventional Pulmonology—Seijo LM, Sterman DH. N Engl J Med 2001;344(10):740–749.

Flow Limitation and Dynamic Hyperinflation During Exercise in COPD Patients After Single Lung Transplantation —Murciano D, Ferretti A, Boczkowski J, Sleiman C, Fournier M, Milic-Emili J. Chest 2000 Nov;118(5):1248-1254

STUDY OBJECTIVE: Using the negative expiratory pressure (NEP) method, we have previously shown that patients receiving single lung transplantation (SLT) for COPD do not exhibit expiratory flow limitation and have little dyspnea at rest. In the present study, we assessed whether SLT patients exhibit flow limitation, overall hyperinflation, and dyspnea during exercise. METHODS: Expiratory flow limitation assessed by the NEP method and inspiratory capacity maneuvers used to determine end-expiratory lung volume (EELV) and end-inspiratory lung volume (EILV) were performed at rest and during symptom-limited incremental cycle exercise in eight SLT patients. RESULTS: At the time of the study, the mean (± SD) FEV₁, IVC, functional residual capacity, and total lung capacity (TLC) amounted to 55 ± 14%, 67 ± 12%, 137 ± 16%, and 110 ± 11% of predicted, respectively. At rest, all patients did not experience expiratory flow limitation and were without dyspnea. At peak exercise, the maximal mechanical power output and maximal oxygen

consumption amounted to 72 ± 20% and 65 ± 8% of predicted, respectively, with a maximal dyspnea Borg score of 6 ± 3. All but one patient exhibited flow limitation and dynamic hyperinflation; the EELV and EILV amounted to 74 ± 5% and 95 ± 9% TLC, respectively. The patient who did not exhibit flow limitation during exercise had the lowest dyspnea score. CONCLUSION: Most SLT patients for COPD exhibit expiratory flow limitation and dynamic hyperinflation during exercise, whereas maximal dyspnea is variable.

Pulmonary Complications Following Lung Resection: A Comprehensive Analysis of Incidence and Possible Risk Factors— Stephan F, Boucheseiche S, Hollande J, Flahault A, Cheffi A, Bazelly B, Bonnet F. Chest 2000 Nov;118(5):1263-1270.

STUDY OBJECTIVES: To assess the incidence and clinical implications of postoperative pulmonary complications (PPCs) after lung resection, and to identify possible associated risk factors. DESIGN: Retrospective study. SETTING: An 885-bed teaching hospital. PATIENTS AND METHODS: We reviewed all patients undergoing lung resection during a 3-year period. The following information was recorded: preoperative

assessment (including pulmonary function tests), clinical parameters, and intraoperative and postoperative events. Pulmonary complications were noted according to a precise definition. The risk of PPCs associated with selected factors was evaluated using multiple logistic regression analysis to estimate odds ratios (ORs) and 95% confidence intervals (CIs). RESULTS: Two hundred sixty-six patients were studied (87 after pneumonectomy, 142 after lobectomy, and 37 after wedge resection). Sixty-eight patients (25%) experienced PPCs, and 20 patients (7.5%) died during the 30 days following the surgical procedure. An American Society of Anesthesiology (ASA) score $\geq$ 3 (OR, 2.11; 95% CI, 1.07 to 4.16; p < 0.02), an operating time > 80 min (OR, 2.08; 95% CI, 1.09 to 3.97; p < 0.02), and the need for postoperative mechanical ventilation > 48 min (OR, 1.96; 95% CI, 1.02 to 3.75; p < 0.04) were independent factors associated with the development of PPCs, which was, in turn, associated with an increased mortality rate and the length of ICU or surgical ward stay. CONCLUSIONS: Our results confirm the relevance of the ASA score in a selected population and stress the importance of the length of the surgical procedure and the need for postoperative mechanical ventilation in the development of PPCs. In addition, preoperative pulmonary function tests do not appear to contribute to the identification of high-risk patients.

The Appropriate Setting of Noninvasive Pressure Support Ventilation in Stable COPD Patients—Vitacca M, Nava S, Confalonieri M, Bianchi L, Porta R, Clini E, Ambrosino N. Chest 2000 Nov;118(5):1286-1293.

STUDY OBJECTIVE: To evaluate the short-term physiologic effects of two settings of nasal pressure-support ventilation (NPSV) in stable COPD patients with chronic hypercapnia. DESIGN: Randomized controlled physiologic study. SETTING: Lung function units and outpatient clinic of two affiliated pulmonary rehabilitation centers. PATIENTS: Twenty-three patients receiving domiciliary nocturnal NPSV for a mean ($\pm$ SD) duration of 31 $\pm$ 20 months. METHODS: Evaluation of arterial blood gases, breathing pattern, respiratory muscles, and dynamic intrinsic positive end-expiratory pressure ($PEEP_{i,dyn}$) during both unassisted and assisted ventilation. Two settings of NPSV were randomly applied for 30 min each: (1) usual setting (U), the setting of NPSV actually used by the individual patient at home; and (2) physiologic setting (PHY), the level of inspiratory pressure support (IPS) and external positive end-expiratory pressure (PEEPe) tailored to patient according to invasive evaluation of respiratory muscular function and mechanics. RESULTS: All patients tolerated NPSV well throughout the procedure. Mean U was IPS, 16 $\pm$ 3 cm H_2O and PEEPe, 3.6 $\pm$ 1.4 cm H_2O; mean PHY was IPS, 15 $\pm$ 3 cm H_2O and PEEPe, 3.1 $\pm$ 1.6 cm H_2O. NPSV was able to significantly (p < 0.01) improve arterial blood gases independent of the setting applied. When compared with spontaneous breathing, both settings induced a significant increase in minute ventilation (p < 0.01). Both settings were able to reduce the diaphragmatic pressure-time product, but the reduction was significantly greater with PHY (by 64%; p < 0.01) than with U (56%; p < 0.05). Eleven of 23 patients (48%) with U and 7 of 23 patients (30%) with PHY showed ineffective efforts (IE); the prevalence of IE (20 $\pm$ 39% vs 6 $\pm$ 11% of their respiratory rate with U and PHY, respectively) was statistically different (p < 0.05). CONCLUSION: In COPD patients with chronic hypercapnia, NPSV is effective in improving arterial blood gases and in unloading inspiratory muscles independent of whether it is set on the basis of patient comfort and improvement in arterial blood gases or tailored to a patient's respiratory muscle effort and mechanics. However, setting of inspiratory assistance and PEEPe by the invasive evaluation of lung mechanics and respiratory muscle function may result in reduction in ineffective inspiratory efforts. These short-term results must be confirmed in the long-term clinical setting.

The Spirometric Efficacy of Once-Daily Dosing with Tiotropium in Stable COPD: A 13-week Multicenter Trial—Casaburi R, Briggs DD Jr, Donohue JF, Serby CW, Menjoge SS, Witek TJ Jr. Chest 2000 Nov; 118(5):1294-302.

STUDY OBJECTIVE: To compare the bronchodilator efficacy and safety of tiotropium and placebo. DESIGN: A 3-month, randomized, double-blind, placebo-controlled, multicenter trial. SETTING: Outpatient. PATIENTS: Four hundred seventy patients with stable COPD (mean FEV_1 = 38.6% predicted). INTERVENTIONS: Tiotropium 18 μg (N = 279) or placebo (N = 191) given once daily via a lactose-based dry-powder inhaler device. MEASUREMENTS AND RESULTS: Spirometry was evaluated on days 1, 8, 50, and 92. Data were expressed as the mean trough (ie, before morning dose; 23 to 24 h after previous dose) and average response observed in the 3 h after the dose was received. Tiotropium produced significant improvement in trough FEV_1 and FVC, averaging 12% greater than baseline on day 8; these improvements were maintained on days 50 and 92. The average postdose FEV_1 was 16% greater than baseline on day 1 and 20% greater than baseline on day 92; FVC was 17% greater than baseline on day 1 and 19% greater than baseline on day 92. Tiotropium was significantly more effective than placebo in both trough and average FEV_1 and FVC response (p < 0.001). These spirometric effects were corroborated by significant improvements in daily morning and evening peak expiratory flow rate, as well as a reduction in "as-needed" albuterol use. Symptoms of wheezing and shortness of breath were significantly less in patients receiving tiotropium, and the physician global assessment noted overall improvements with those treated with tiotropium relative to placebo. The most common reported adverse event after tiotropium was dry mouth (9.3% vs 1.6% relative to placebo; p < 0.05). CONCLUSIONS: These data demonstrate that tiotropium is a safe and effective once-daily anticholinergic bronchodilator and should prove useful as first-line maintenance therapy in COPD.

Appropriateness of Domiciliary Oxygen Delivery—Guyatt GH, McKim DA, Austin P, Bryan R, Norgren J, Weaver B, Goldstein RS. Chest 2000 Nov;118(5):1303-1308.

OBJECTIVE: Almost every country in the developed world has a domiciliary oxygen program. Whether recipients meet program criteria has not been rigorously studied. DESIGN: Cross-sectional survey. PARTICIPANTS: Two hundred thirty-seven patients receiving domiciliary oxygen in the Ontario Ministry of Health Home Oxygen Program (HOP). METHODS: A respiratory therapist visited the patients' homes and administered questionnaires, obtained resting arterial blood gas measurements, and conducted a standardized home exercise test while monitoring oxygen saturation using an oximeter. Measures of outcome: We evaluated the extent to which patients met HOP criteria that are based on the inclusion criteria of randomized trials showing the life-prolonging effects of domiciliary oxygen. We also assessed the extent to which the patients' oxygen prescription was consistent with the results of rest and exercise testing. RESULTS: Ninety-six of 237 participants (40.5%; 95% confidence interval, 34.3 to 46.8) did not meet criteria for home oxygen. Patients aged < 70 years were more likely to meet criteria (71 of 105 patients, 67.9%) than those > 70 years old (70 of 132 patients; 53.0%). The proportion of patients meeting criteria was similar whether the referring physician was a specialist (71 of 112 patients; 62.5%) or a primary-care physician (69 of 123 patients; 56.1%). A very important health benefit from oxygen was identified among 82% of those who met criteria and 88% of those who did not. Patients received higher flow rates than our criteria suggested were appropriate. Agreement between the independently assessed oxygen prescription at rest and the patients' report of oxygen use was extremely poor (chance-corrected agreement κ, 0.17), as was agreement concerning optimal exercise flow rates (κ, 0.26). CONCLUSIONS: Current procedures for administration and reimbursement of home oxygen result in a large proportion of recipients not meeting criteria, as well as the prescription of excessive oxygen flow rates. These results are likely to apply to many jurisdictions and suggest a large potential for more efficient resource allocation.

Asthmatic Subjects Symptomatically Worse at Work: Prevalence and Characterization Among a General Asthma Clinic Population—Tarlo SM, Leung K, Broder I, Silverman F, Holness DL. Chest 2000 Nov,118(5):1309-1314.

STUDY OBJECTIVES: To assess the prevalence of a historical occupational component to asthma in an adult asthma clinic and to compare characteristics of asthmatic subjects with and without work-attributed symptoms. DESIGN: A retrospective review of data obtained from a physician-administered questionnaire, answers to which were obtained at the initial patient visit of asthmatic subjects, and which included specific questions regarding the relationship of work to symptoms. Chart review data were used to supplement information on workplace exposures and investigations. SETTING: A university-based secondary- and tertiary-referral asthma clinic. Patients: Seven hundred thirty-one adult asthmatic subjects who were referred for assessment and management of asthma. INTERVENTIONS: Statistical analyses of asthmatic subjects with and without work-attributed symptoms and a determination, from chart review, of the likelihood of causes for symptomatic worsening of asthma at work. MEASUREMENTS AND RESULTS: Sixty percent of the patients (435) had adult onset of asthma, among whom 310 patients (71%) were employed at the time of their visit. Fifty-one patients reported their asthma to be worse at work (ie, 16% of adult-onset working asthmatic subjects). Sixteen of these patients (31%) had likely or possible sensitizer-induced occupational asthma (OA), and 49% likely had aggravation of underlying asthma. The other 20% of patients had possible OA or aggravation of underlying asthma at work. CONCLUSIONS: Adult-onset asthmatic subjects commonly report a worsening of asthma at work, more commonly on the basis of likely aggravation of underlying asthma than on the basis of likely or possible OA.

Effects of Weight Loss on Peak Flow Variability, Airways Obstruction, and Lung Volumes in Obese Patients with Asthma—Hakala K, Stenius-Aarniala B, Sovijarvi A. Chest 2000 Nov,118(5):1315-1321.

STUDY OBJECTIVES: To clarify the pathophysiologic features of the relation between asthma and obesity, we measured the effects of weight reduction on peak expiratory flow (PEF) variability and airways obstruction, compared to simultaneous changes in lung volumes and ventilatory mechanics in obese patients with stable asthma. METHODS: Fourteen obese asthma patients (11 women and 3 men; aged 25 to 62 years) were studied before and after a very-low-calorie-diet period of 8 weeks. PEF variability was determined as diurnal and day-to-day variations. FEV_1 and maximal expiratory flow values were measured with a flow-volume spirometer. Lung volumes, airways resistance (R_{aw}), and specific airways conductance were measured using a constant-volume body plethysmograph. Minute ventilation was monitored in patients in supine and standing positions. RESULTS: As patients decreased their body mass index (SD) from 37.2 (3.7) to 32.1 (4.2) kg/m² (p < 0.001), diurnal PEF variation declined from 5.5% (2.4) to 4.5% (1.5) (p = 0.01), and day-to-day variation declined from 5.3% (2.6) to 3.1% (1.3) (p < 0.005). The mean morning PEF, FEV_1, and FVC increased after weight loss (p = 0.001, p < 0.005, and p < 0.05, respectively). Flow rate at the middle part of FVC (FEF_{25-75}) increased even when related to lung volumes (FEF_{25-75}/FVC, p < 0.05). Functional residual capacity and expiratory reserve volume were significantly higher after weight loss (p < 0.05 and p < 0.005, respectively). A significant reduction in R_{aw} was found (p < 0.01). Resting minute ventilation decreased after weight loss (p = 0.01). CONCLUSION: Weight loss reduces airways obstruction as well as PEF variability in obese patients with asthma. The results suggest that obese

patients benefit from weight loss by improved pulmonary mechanics and a better control of airways obstruction.

Inspiratory Effort Sensation to Added Resistive Loading in Patients with Obstructive Sleep Apnea — Tun Y, Hida W, Okabe S, Kikuchi Y, Kurosawa H, Tabata M, Shirato K. Chest 2000 Nov;118(5):1332-1338.

STUDY OBJECTIVES: Repeated episodes of upper airway occlusion are the main characteristics of patients with obstructive sleep apnea (OSA) during sleep. It has been reported that an impairment in the sensation of detection and a depression of ventilatory compensation to added load could be observed in such patients. In this study, we examined patients with OSA to evaluate the inspiratory effort sensation (IES), ventilation, and mouth occlusion pressures during added resistive loading while awake and to determine whether they can be reversed by nasal continuous positive airway pressure (CPAP) treatment. DESIGN: A hospital-based case-control study. SETTING: A sleep laboratory of a medical unit in Japan. SUBJECTS: Seventeen patients with moderate to severe OSA and 10 control subjects were included in this study. MEASUREMENTS: All patients with OSA had undergone standard nocturnal polysomnography. Patients with OSA and control subjects were evaluated for IES measured by a modified Borg score, ventilation, and mouth occlusion pressure during control and inspiratory resistive loaded breathing. These tests were repeated in all patients with OSA after 2 weeks of nasal CPAP treatment. RESULTS: IES to inspiratory resistive loading was lower in patients with OSA than in control subjects. There were no differences in ventilation and mouth occlusion pressure between patients and control subjects during loaded breathing. After 2 weeks of nasal CPAP, the decreased IES was increased in patients with OSA. CONCLUSION: In patients with OSA, the decreased IES to inspiratory resistive loaded breathing is reversible with nasal CPAP. This could be one additional benefit of nasal CPAP in the treatment of OSA.

Prevention of Pulmonary Morbidity for Patients with Neuromuscular Disease — Tzeng AC, Bach JR. Chest 2000 Nov;118(5):1390-1396.

STUDY OBJECTIVE: To evaluate the effects of a respiratory muscle and protocol on hospitalization rates for respiratory complications of neuromuscular disease. DESIGN: A retrospective cohort study. METHODS: A home protocol was developed in which oxyhemoglobin desaturation was prevented or reversed by the use of noninvasive intermittent positive-pressure ventilation and manually and mechanically assisted coughing as needed. The patients who had more than one episode of respiratory failure before having access to the protocol were considered to have had preprotocol periods (group 1). Other patients were given access to the protocol when their assisted peak cough flows decreased to < 270 L/min before any episodes of respiratory distress (group 2). The number of hospitalizations and days hospitalized were compared longitudinally for preprotocol and protocol access periods (group 1). In addition, avoided hospitalizations were identified as "episodes" of need for continuous ventilatory support and desaturations reversed by assisted coughing that were managed at home. Data were segregated by access to protocol and by extent of baseline ventilator use. RESULTS: Of the 47 group 1 patients with preprotocol periods who have subsequently had episodes, 10 had episodes before requiring ongoing ventilator use. They had 1.06 ± 0.84 preprotocol hospitalizations per year per patient and 20.76 ± 36.01 hospitalization days per year per patient over 3.42 ± 3.36 years per patient vs 0.03 ± 0.11 hospitalizations per year per patient and 0.06 ± 0.20 hospitalization days per year per patient with protocol use over 1.94 ± 0.74 years per patient. Of these 47 group 1 patients, 33 eventually required part-time ventilatory aid and, using the protocol as needed, had 0.08 ± 0.17 hospitalizations per year per patient and 1.43 ± 3.71 hospitalization days per year per patient over 3.91 ± 3.50 years per patient, as opposed to 1.40 ± 1.96 hospitalizations per year per patient and 20.14 ± 41.15 hospitalization days per year per patient preprotocol and preventilator use over 5.89 ± 6.89 years per patient. Twelve patients in group 1 eventually required continuous noninvasive ventilation and, using the protocol as needed, had 0.07 ± 0.11 hospitalizations per year per patient and 0.39 ± 0.73 hospitalization days per year per patient over 5.35 ± 5.10 years per patient by comparison with 0.97 ± 0.74 hospitalizations per year per patient and 10.39 ± 8.66 hospitalization days per year per patient over 2.18 ± 1.91 years per patient preprotocol and preventilator use. For the 94 patients overall when having access to the protocol, 1.02 ± 0.99 hospitalizations per year per patient were avoided by 14 patients before requiring ongoing ventilator use over 4.82 ± 1.61 years, 0.99 ± 1.12 hospitalizations per year per patient were avoided by 73 part-time ventilator users over 3.21 ± 3.15 years, and 0.80 ± 0.85 hospitalizations per year per patient were avoided by 31 full-time ventilator users over 4.78 ± 4.88 years. All preprotocol and protocol rate comparisons were statistically significant at p < 0.004. CONCLUSION: Patients have significantly fewer hospitalizations per year and days per year when using the protocol as needed than without the protocol. The use of inspiratory and expiratory aids can significantly decrease hospitalization rates for respiratory complications of neuromuscular disease.

Different Response to Doubling and Fourfold Dose Increases in Methacholine Provocation Tests in Healthy Subjects — Sundblad BM, Malmberg P, Larsson K. Chest 2000 Nov;118(5):1371-1377.

RATIONALE: In a modified methacholine provocation test that was used to study changes in airway responsiveness to occupational irritants or sensitizers in healthy subjects, two protocols were used: a long protocol (doubling methacholine concentrations between dose steps) or a short protocol (fourfold increases in concentration). This modified methacholine provocation allows measurements of the provocative dose causing 20% decrease in FEV_1 (PD_{20}) in a high proportion of a normal population. METHODS: The distribution of PD_{20} was investigated in healthy nonatopic men without history of allergy or asthma symptoms using the long protocol (n = 101) or the short protocol (n = 309). In addition, 30 healthy subjects underwent methacholine provocation tests using both protocols. RESULTS: PD_{20} was defined in 79% of subjects with the long protocol and in 48% of subjects with the short protocol. The provocative concentration of methacholine causing a 20% decline in FEV_1 (PC_{20}) and PD_{20} were significantly lower using the long protocol: long-protocol PC_{20} (median [25th to 75th percentile]), 19.9 mg/mL (3.9 to > 32 mg/mL) compared with short-protocol PC_{20}, > 32 mg/mL (8.7 to > 32 mg/mL, p < 0.0001); long-protocol PD_{20}, 4.2 mg (1.6 to 20 mg) compared with short-protocol PD_{20}, > 13.7 (2.6 to > 13.7 mg, p = 0.006). The differences in PD_{20} using short and long protocols were confirmed in a randomized trial of 30 healthy subjects tested with both protocols. CONCLUSION: Using doubling concentrations, PC_{20} and PD_{20} could be defined in a higher proportion of healthy subjects than a protocol using fourfold dose increases. Furthermore, the doubling protocol results in a PD_{20} estimate that is less than half the value obtained when using a protocol with fourfold concentrations between dose steps. The difference remains, whether the methacholine effect is regarded as cumulative or noncumulative. The explanation for the difference between the protocols is unclear.

Routine Pulse Oximetry During Methacholine Challenges Is Unnecessary for Safety — Cockcroft DW, Hurst TS, Marciniuk DD, Cotton DJ, Latramboise KF, Nagpal AK, Skomro RP. Chest 2000 Nov;118(5):1378-1381.

BACKGROUND: Methacholine-induced bronchoconstriction is associated with significant hypoxemia, which can be assessed noninvasively by transcutaneous oxygen tension and pulse oximetry. OBJECTIVES: To assess the value of the monitoring of finger pulse oximetry during routine methacholine challenges in a clinical pulmonary function laboratory with regard to both safety and the possibility that a significant fall in oxygen

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saturation as measured by pulse oximetry (S_{pO_2}) might be a useful surrogate for determining the response to methacholine. METHODS: Two hundred consecutive patients undergoing diagnostic methacholine challenges in the pulmonary function laboratory of a tertiary-care, university-based referral hospital were studied. Methacholine challenges were performed by the standardized 2-min tidal breathing technique, and the ΔFEV_1 was calculated from the lowest postsaline solution inhalation to the lowest postmethacholine inhalation value. S_{pO_2} was measured immediately prior to each spirogram, and the ΔS_{pO_2} was measured from the lowest postsaline solution inhalation value to the lowest postmethacholine inhalation value. We examined the data for safety (ie, any S_{pO_2} value < 90). Based on previous reports, we used a ΔS_{pO_2} of > 3 as significant and looked at the sensitivity, specificity, and positive and negative predictive values for $\Delta S_{pO_2} \geq 3$ vis-a-vis a fall in FEV_1 of $\geq 15\%$. RESULTS: There were 119 nonresponders ($\Delta FEV_1 < 15\%$) and 81 responders. The baseline FEV_1 percent predicted was slightly but significantly lower in the responders (responders [± SD], 91.6 ± 15%; nonresponders, 96.4 ± 14%; p < 0.05). ΔS_{pO_2} was 3.1 ± 1.6 in the responders and 1.6 ± 1.8 in the nonresponders (p < 0.001). There was a single recording in one patient of $S_{pO_2} < 90$ (88). A $\Delta S_{pO_2} \geq 3$ had a sensitivity of 68%, a specificity of 73%, a positive predictive value of 63%, and negative predictive value of 77% for a fall in $FEV_1 \geq 15\%$. CONCLUSIONS: Pulse oximetry is not routinely useful for safety monitoring during methacholine challenge. ΔS_{pO_2} is not helpful in predicting a positive spirometric response to methacholine. However, the negative predictive value is adequate to allow the ΔS_{pO_2} to be used as an adjunct in assessing a negative result of a methacholine test in patients who have difficulty performing spirometry.

A Meta-Analysis of Prospective Trials Comparing Percutaneous and Surgical Tracheostomy in Critically Ill Patients—Freeman BD, Isabella K, Lin N, Buchman TG. Chest 2000 Nov;118(5):1412-1418.

STUDY OBJECTIVES: Tracheostomy is one of the most commonly performed procedures in the patient receiving long-term mechanical ventilation. While percutaneous dilational tracheostomy (PDT) is becoming increasingly utilized as an alternative to conventional surgical tracheostomy, most literature evaluating these two techniques is neither prospective nor controlled. We performed a meta-analysis of available prospective controlled studies comparing PDT and surgical tracheostomy in critically ill patients to more fully understand the relative benefits and risks of these two procedures in this population. DESIGN: Meta-analysis using Mantel-Haenszel fixed effect model. INTERVENTIONS: We performed searches of MEDLINE, Current Contents, Best Evidence, Cochrane, and HealthSTAR databases from 1985 to present to identify prospective controlled studies comparing PDT and surgical tracheostomy in critically ill patients. After establishing clinical and statistical homogeneity (Q statistic), studies were analyzed by a Mantel-Haenszel fixed effect model. For each clinical end point examined, PDT and surgical tracheostomy were compared by calculating either absolute differences or odds ratios (ORs) with 95% confidence intervals (CIs) for continuous or discrete variables, respectively. Measurements and results: We pooled data from five studies (236 patients) satisfying our search criteria to analyze eight clinical end points. Operative time was shorter for PDT than surgical tracheostomy, absolute difference with 95% CI, 9.84 min (7.83 to 10.85 min). There was no difference comparing PDT and surgical tracheostomy with respect to overall operative complication rates (OR with 95% CI, 0.732 [0.05 to 9.37]). However, relative to surgical tracheostomy, PDT was associated with less perioperative bleeding (OR with 95% CI, 0.14 [0.02 to 0.39]), a lower overall postoperative complication rate (OR with 95% CI, 0.14 [0.07 to 0.29]), as well as a lower postoperative incidence of bleeding (OR with 95% CI, 0.39 [0.17 to 0.88]), and stomal infection (OR with 95% CI, 0.02 [0.01 to 0.07]). No difference was identified in days intubated prior to tracheostomy (absolute difference with 95% CI, 0.16 days [−0.09 to 1.22 days]), overall

procedure-related complications (OR with 95% CI, 0.73 [0.06 to 9.37]), or death (OR with 95% CI, 0.63 [0.18 to 2.20]) comparing these two techniques. CONCLUSIONS: Despite its popularity, there are currently only a limited number of small studies prospectively evaluating PDT and surgical tracheostomy. Our meta-analysis of these studies suggests potential advantages of PDT relative to surgical tracheostomy, including ease of performance, and lower incidence of peristomal bleeding and postoperative infection. If confirmed by additional, adequately powered prospective trials, these findings support PDT as the procedure of choice for the establishment of elective tracheostomy in the appropriately selected critically ill patient.

End-of-Life Care in the ICU: Treatments Provided when Life Support Was or Was Not Withdrawn—Hall RI, Rocker GM. Chest 2000 Nov;118(5):1424-1430.

STUDY OBJECTIVE: To compare and contrast use of technology, pharmacology, and physician variability in end-of-life care of ICU patients dying with or without active life support. DESIGN: Retrospective cohort study. SETTING: Two medical-surgical tertiary-care ICUs in a Canadian regional referral teaching hospital. PARTICIPANTS: One hundred seventy-four patients who died between July 1, 1996, and June 30, 1997. INTERVENTION: Data abstraction from medical records. RESULTS: Patients in whom life support was withheld or withdrawn (138 of 174, 79%) were older (65 ± 16 years vs 55 ± 18 years; p < 0.05 [mean ± SD]). Once the decision to withdraw life support was made, death occurred in 4.3 h (2.1 to 6.5 h; mean [95% confidence interval]). Patients who had active life support treatment until death received more support measures including inotropic agents (36 of 36 vs 21 of 138; p < 0.05), dialysis (4 of 36 vs 2 of 138; p < 0.05), and mechanical ventilation at the time of death (36 of 36 vs 81 of 138; p < 0.05). Physician differences (> 10-fold) were detected for prescribed doses of morphine and sedative agents whether or not life support was withheld or withdrawn. The median cumulative dose of morphine prescribed during the final 12 h was larger (fivefold) in patients undergoing withdrawal of life support. No documented discussion of life support withdrawal was noted in one case. In the remaining patients, the 10 staff physicians were documented to be involved in 77% (range, 54 to 94%) of the end-of-life discussions. CONCLUSIONS: Differences were evident in technologic and pharmacologic support and in physician prescribing habits in patients for whom life support was or was not withheld or withdrawn. Substantial variability was noted in physician documentation of physician-family interactions surrounding the withdrawal of life support.

Managing Life-Threatening Hemoptysis: Has Anything Really Changed? Haponik EF, Fein A, Chin R. Chest 2000 Nov;118(5):1431-1435.

STUDY OBJECTIVES: To delineate current chest clinicians' approaches to the management of patients with life-threatening hemoptysis. DESIGN: Survey during a computer-assisted interactive continuing medical education presentation. SETTING: The 1998 American College of Chest Physicians (ACCP) Annual Scientific Assembly. PARTICIPANTS: Chest clinicians attending the respiratory emergency symposium. RESULTS: Most clinicians (86%) had cared for patients with life-threatening hemoptysis, and 28% had cared for patients with fatal events during the previous year. Those clinicians favored management in the ICU setting (95%) with early endotracheal intubation (85%), and they tended to use a large-bore, single-lumen endotracheal tube (57%). The majority (64%) favored the early performance of diagnostic bronchoscopy during the first 24 h. Most clinicians (79%) used the flexible instrument, a higher frequency than respondents at a similar symposium on hemoptysis at the 1988 ACCP meeting (48%; p < 0.0001). Most current clinicians (77%) had experience with endobronchial measures to control bleeding, but few (14%) found them to be consistently worthwhile. Chest CT scanning was

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often helpful in diagnosis (55%). In their management of bleeding, half of these clinicians favored the use of interventional angiography, even in operable patients, which is a substantial change from 1988 when 23% had favored this approach (p < 0.0001). CONCLUSIONS: During the past decade, life-threatening hemoptysis has remained an important problem. Flexible bronchoscopy and interventional angiography have become increasingly established, more widely accepted approaches to patient care.

A Morphologic Study of Long-Term Retention of Fluorocarbon After Liquid Ventilation — Hood CI, Modell JH. Chest 2000 Nov;118(5):1436-1440

STUDY OBJECTIVES: To determine how long perfluorinated hydrocarbons remain in the lung after they are used for lung ventilation in dogs, and to determine if residual perfluorinated hydrocarbons cause structural alteration or an inflammatory reaction of the lung. DESIGN: Adult dogs were anesthetized and received ventilation with oxygenated perfluorinated hydrocarbon liquid. Morphologic studies of tissue from the lungs of these dogs were performed at intervals of a few minutes to 10 years after reconversion to breathing gas. SETTING: University College of Medicine. PARTICIPANTS: Adult mongrel and beagle dogs. INTERVENTIONS: Anesthetized adult dogs breathed oxygenated liquid fluorocarbons for 1 h and then were reconverted to breathing air. Three fluorocarbons, FX-80 ($C_8F_{16}O$, 3M Company; St Paul, MN), Caroxin-D ($C_{10}F_{23}O_3$; P-1D, Allied Chemical Company; Morristown, NJ), and Caroxin-F ($C_9F_{20}O$; P-12F, Allied Chemical Company), were used. Morphologic studies of the lungs of these animals were performed immediately after restoration of air breathing and at intervals for up to 10 years. Not all animals were studied at each time interval. MEASUREMENTS AND RESULTS: A transient, acute inflammatory reaction was followed by a massive influx of macrophages, which were at first intra-alveolar and later interstitial, especially around vessels and bronchioles. Fluorocarbons remained in the lung in diminishing amounts for at least 5 years, as evidenced by persistent vacuolated macrophages in the alveoli, interstitium, and hilar lymph nodes; fluorocarbon was also detected in these tissues by chemical assays. In no case was there fibrosis or any other structural alteration associated with the residual fluorocarbon, which suggests that it was inert. At 10 years, no evidence of residual fluorocarbon was seen morphologically.

Nasal Continuous Positive Airway Pressure Devices Do Not Maintain the Set Pressure Dynamically when Tested Under Simulated Clinical Conditions — Bacon JP, Farney RJ, Jensen RL, Walker JM, Cloward TV. Chest 2000 Nov;118(5):1441-1449.

STUDY OBJECTIVES: Nasal continuous positive airway pressure (CPAP) is standard therapy for obstructive sleep apnea syndrome. The effective nasal mask pressure may be adversely affected by factors that increase system resistance (eg, long tubing and/or water condensation) and by dynamic variables (breathing frequency [f] and tidal volume [V_T]). The present study was conducted in order to assess the performance of CPAP machines throughout a range of simulated clinical conditions. DESIGN: Four currently used CPAP machines were tested at settings of 5, 10, 15, and 20 cm H_2O using a pulmonary waveform generator to produce V_Ts of 0.4, 0.8, and 1.2 L at frequencies of 10, 20, and 30 breaths/min. Machines were tested under five conditions: 6-foot and 12-foot tubing, with and without an in-line humidifier, and 12-foot tubing with humidifier and water condensation. MEASUREMENTS: Maximum and minimum mask pressure measurements were obtained during five respiratory cycles for each dynamic variable under each of the five conditions and CPAP settings (180 experiments on each of four CPAP models). RESULTS: Using typical clinical parameters (V_T, 0.4 L and 0.8 L; f, 10 breaths/min and 20 breaths/min; and CPAP, 5 to 15 cm H_2O), mask pressure consistently varied above and below the set point when additional tubing and/or a humidifier were added to the system (0.7

to 2.9 cm H_2O below and 0.5 to 1.0 cm H_2O above the set pressure). Water condensation caused large pressure deviations (inspiratory pressure ranged from 3.5 to 5.6 cm H_2O below set pressure, and expiratory pressure ranged from 0.7 to 3.5 cm H_2O above set pressure). CONCLUSIONS: Therapy and compliance could be adversely affected because some CPAP machines in current use do not maintain constant continuous mask pressure when tested using simulated conditions, especially when water condenses in the tubing.

Work of Breathing During Spontaneous Ventilation in Anesthetized Children: A Comparative Study Among the Face Mask, Laryngeal Mask Airway and Endotracheal Tube — Keidan I, Fine GF, Kagawa T, Schneck FX, Motoyama EK. Anesth Analg 2000 Dec;91(6):1381-1388.

Work of breathing (WOB) increases during general anesthesia in adults, but such information has been limited in pediatric patients. We studied WOB in 24 healthy children (mean age 2 ± 1.9 yrs), during elective urogenital surgery under 1 minimum alveolar anesthetic concentration halothane-nitrous oxide anesthesia with a caudal block while breathing spontaneously. WOB was measured with an esophageal balloon, miniature flowmeter, and a computerized (Bicore) system. In each patient, WOB was computed under four conditions: a mask without oral airway (-AW), a mask with oral airway (+AW), a laryngeal mask airway (LMA), and an endotracheal tube (ETT). With each apparatus WOB was studied both with continuous positive airway pressure (CPAP) (5-6 cm H_2O) and without CPAP (or zero end-expiratory pressure [ZEEP]). Under ZEEP, WOB (g · cm/kg) among the four apparatus were (mean ± SEM): mask (-AW) (64 ± 19.2) > mask (+AW) (44 ± 17.2), LMA (42 ± 15.6) > ETT (25.4 ± 12.4) (p < 0.05). WOB with CPAP significantly (p < 0.05) decreased from WOB with ZEEP in three groups (mask [-AW], mask [+AW], and LMA), but not in the ETT group. Tidal volume (both ZEEP and CPAP) and end-tidal P_{CO_2} (with CPAP only) were significantly (p < 0.05) decreased only in the ETT group, whereas no significant difference was found in respiratory rate or minute volume among the four airway apparatus groups, either with or without CPAP. The reduction in WOB, when breathing through ETT was primarily attributable to decreases in tidal volume and volume work. The finding that WOB decreases with CPAP in all groups except for the ETT group suggests that the decrease is a result of improved patency of the upper airway rather than of increases in functional residual capacity and lung compliance. Implications: We studied work of breathing (WOB) measured with four airway devices, with and without application of continuous positive airway pressure (CPAP). Laryngeal mask airway and mask with oral airway decrease WOB compared with mask alone. CPAP decreases WOB with all devices except the endotracheal tube. Increased WOB appears mostly because of soft tissue upper airway obstruction.

Auditory Steady-State Response and Bispectral Index for Assessing Level of Consciousness During Propofol Sedation and Hypnosis — Bonhomme V, Plourde G, Meuret P, Fiset P, Backman SB. Anesth Analg 2000 Dec;91(6):1398-1403.

We assessed the effect of propofol on the auditory steady-state response (ASSR), bispectral (BIS) index, and level of consciousness in two experiments. In Experiment 1, propofol was infused in 11 subjects to obtain effect-site concentrations of 1, 2, 3, and 4 µg/mL. The ASSR and BIS index were recorded during baseline and at each concentration. The ASSR was evoked by monaural stimuli. Propofol caused a concentration-dependent decrease of the ASSR and BIS index values (r^2 0.76 and 0.93, respectively, p < 0.0001). The prediction probability for loss of consciousness was 0.89, 0.96, and 0.94 for ASSR, BIS, and arterial blood concentration of propofol, respectively. In Experiment 2, we compared the effects of binaural versus monaural stimulus delivery on the ASSR in six subjects during awake baseline and propofol-induced unconsciousness. During baseline, the ASSR amplitude with binaural stimulation

(0.47 ± 0.13 μV, mean ± SD) was significantly (p = 0.002) larger than with monaural stimulation (0.35 ± 0.11 μV). During unconsciousness, the amplitude was 0.09 ± 0.09 μV with monaural and 0.06 ± 0.04 μV with binaural stimulation (NS). The prediction probability for loss of consciousness was 0.97 (0.04 SE) for monaural and 1.00 (0.00 SE) for binaural delivery. We conclude that the ASSR and BIS index are attenuated in a concentration-dependent manner by propofol and provide a useful measure of its sedative and hypnotic effect. BIS was easier to use and slightly more sensitive. The ASSR should be recorded with binaural stimulation. The ASSR and BIS index are both useful for assessing the level of consciousness during sedation and hypnosis with propofol. However, the BIS index was simpler to use and provided a more sensitive measure of sedation. Implications: We have compared two methods for predicting whether the amount of propofol given to a human subject is sufficient to cause unconsciousness, defined as failure to respond to a simple verbal command. The two methods studied are the auditory steady-state response, which measures the electrical response of the brain to sound, and the bispectral index, which is a number derived from the electroencephalogram. The results showed that both methods are very good predictors of the level of consciousness, however, bispectral was easier to use.

Aspiration in Transtracheal Oxygen Insufflation with Different Insufflation Flow Rates During Cardiopulmonary Resuscitation in Dogs—Jawan B, Cheung HK, Chong ZK, Poon YY, Cheng YF, Chen HS, et al. Anesth Analg 2000 Dec;91(6):1431-1435.

We investigated whether transtracheal insufflation of oxygen with different insufflation flow rates protects against aspiration of gastric contents during cardiopulmonary resuscitation (CPR). Its ventilation and oxygenation effects were also evaluated. Cardiac arrest was induced in anesthetized and paralyzed 18 mongrel dogs. Chest compression using an automatic thumper was performed while the dogs randomly received no mechanical ventilation (Group I, n = 6) or were transtracheally insufflated with 4 L/min oxygen (Group II, n = 6) or 10 L/min oxygen (Group III, n = 6). Blood samples were drawn every 5 min for 20 min for blood gas analysis, the mouths of the dogs were then filled with 70 mL mixed barium, and 10 min after chest compression, chest radiographs were taken to evaluate the incidence of pulmonary aspiration. Results showed that pulmonary aspiration occurred in all dogs of Group I and three of the six dogs in Group II, whereas dogs in Group III were free from pulmonary aspiration. Both transtracheal oxygen insufflation groups maintained oxygen saturation significantly better than Group I, but mild hypercapnia was observed in all groups after 20 min of CPR. We conclude that transtracheal oxygen insufflation, but not chest compression alone, was able to maintain oxygenation for 20 min during CPR in dogs with cardiac arrest. Mild hypercapnia was noted in all groups. Chest compression alone caused pulmonary aspiration, whereas insufflation of 10 L O_2/min provided better protection against pulmonary aspiration than that of 4 L O_2/min. Implications: In case of difficult airway during cardiopulmonary resuscitation, insertion of an I.V. catheter through the trachea is easy, and insufflation of 10 L/min of oxygen through the needle can not only maintain the oxygenation but also prevent aspiration.

Respiratory Efficacy of Subglottic Low-Frequency, Subglottic Combined-Frequency, and Supraglottic Combined-Frequency Jet Ventilation During Microlaryngeal Surgery—Bacher A, Lang T, Weber J, Aloy A. Anesth Analg 2000 Dec;91(6):1506-1512.

We tested the respiratory efficacy of different jet ventilation techniques (subglottic low-frequency versus subglottic combined-frequency and subglottic combined-frequency versus supraglottic combined frequency) in patients undergoing microlaryngeal surgery. The P_{aCO_2} and the quotient of arterial oxygen tension (P_{aO_2}) over I_{O_2} were measured. After anesthetic induction (propofol, remifentanil, vecuronium), an endotracheal

Mon-Jet catheter (Xomed, Jacksonville, FL) for subglottic jet ventilation and a laryngoscope for supraglottic jet ventilation (Carl Reiner GmbH Vienna, Austria) were inserted. In Group 1 (n = 18), subglottic low frequency (15 breaths/min), combined frequency (600 and 15 breaths/min), and low-frequency jet ventilation was subsequently performed (15 min each). In Group 2 (n = 19), the sequence was supraglottic, subglottic, and supraglottic combined frequency jet ventilation. The driving pressures were initially adjusted to achieve normocapnia and were not changed during the entire study period. The I_{O_2} was measured endotracheally. The Wilcoxon's signed rank test was applied. In Group 1, P_{aCO_2} and P_{aO_2}/I_{O_2} improved significantly after switching from subglottic low-frequency to subglottic combined frequency jet ventilation (P_{aCO_2}, from 46.6 ± 8.3 to 42.1 ± 8.1 mm Hg; P_{aO_2}/I_{O_2}, from 311 ± 144 to 361 ± 141 mm Hg; p < 0.05). In Group 2, P_{aCO_2} increased and P_{aO_2}/I_{O_2} decreased significantly after switching from supraglottic to subglottic combined frequency jet ventilation (P_{aCO_2}, from 39.4 ± 7.1 to 45.9 ± 7.5 mm Hg; P_{aO_2}/I_{O_2}, from 415 ± 114 to 351 ± 129 mm Hg; p < 0.05). We conclude that subglottic combined-frequency jet ventilation is less effective than supraglottic combined-frequency ventilation, but more effective than subglottic low-frequency jet ventilation. Implications: The combination of high and low respiratory frequencies (600 and 15 breaths/min) improves pulmonary gas exchange during subglottic jet ventilation via an endotracheal catheter. However, subglottic combined frequency jet ventilation is less effective than supraglottic combined-frequency jet ventilation via a jet ventilation laryngoscope.

The Effects of the Reverse Trendelenburg Position on Respiratory Mechanics and Blood Gases in Morbidly Obese Patients During Bariatric Surgery—Perilli V, Sollazzi L, Bozza P, Modesti C, Chierichini A, Tacchino RM, Ranieri R. Anesth Analg 2000 Dec;91(6):1520-1525.

Anesthesia adversely affects respiratory function, particularly in morbidly obese patients. Although many studies have been performed to determine the optimal ventilatory settings in these patients, this question has not been answered. The aim of this study was to evaluate the effect of reverse Trendelenburg position (RTP) on gas exchange and respiratory mechanics in 15 obese patients undergoing biliopancreatic diversion. A standardized anesthetic regimen was used and patients were examined at standard times. 1) after tracheal intubation, 2) after laparotomy, 3) after positioning of subcostal retractors, 4) with retractors in RTP. The measurements of respiratory mechanics were repeated for a wide range of tidal volumes by using the technique of rapid occlusion during constant flow inflation. We noted a wide alveolar-arterial oxygen difference [P(A-a)O_2] in all patients, particularly during Phase 3. When the patients were placed in RTP, P(A-a)O_2 showed a significant improvement and a return toward baseline values. As for mechanics, total respiratory system compliance was significantly higher in RTP than in the other phases. In conclusion, our data suggest that RTP is an appropriate intraoperative posture for obese subjects because it causes minimal arterial blood pressure changes and improves oxygenation. Implications: The aim of the study was to assess whether the reverse Trendelenburg position could improve pulmonary gas exchange in obese patients undergoing abdominal surgical procedures. Our work may have a clinical value because few studies deal with this issue.

Characterization of a Microprocessor-Controlled Tubular Multiple Metered Dose Inhaler Aerosol Generator for Inhalation Exposures of Pharmaceuticals—Rothenberg SJ, Barnett JF, Dearlove GE, Parker RM, Ball DJ, Brady JT, et al. J Aerosol Med 2000;13(3):157-168.

A microprocessor-controlled tubular multiple metered dose inhaler (MDI) aerosol generator was constructed for the delivery of pharmaceutical aerosols to inhalation chambers. The MDIs were mounted in four cassettes containing one to four MDIs on a stepped end plate. The MDIs in each cassette were pneumatically activated at intervals that were con-

trolled by the microprocessor. The cassettes permitted easy replacement of each set of MDIs with a fresh set of MDIs whenever necessary. Aerosol concentration was controlled by varying the number of active MDIs in each cassette and the frequency of activations per minute of each row. Aerosol from the MDIs flowed along the long axis of the tube, which provided a path length sufficient to diminish impaction losses. Using a light-scattering device to monitor the aerosol concentration, the pulsatile output from the MDIs in the cassettes was demonstrated to be adequately damped out provided that the dilution/mixing/aging chamber exceeded 3 ft in length. The tube diameter selected was the minimum compatible with mounting the required number of MDIs so that the linear velocity of the aerosol was adequate to efficiently transport the aerosol out of the dilution chamber. Aerosol concentration and particle size data were recorded for a nose-only rodent exposure chamber. Reproducible aerosol concentrations ranging from 0.03 to 0.6 mg/L were generated. Particle sizes ranged from 2- to 3-mum mass median aerodynamic diameter. Thus, the aerosol generated was within the size range suitable for inhalation exposures.

An Investigation of the Solubility of Various Compounds in the Hydrofluoroalkane Propellants and Possible Model Liquid Propellants—Dickinson PA, Seville PC, McHale H, Perkins NC, Taylor G. J Aerosol Med 2000;13(3):179-186.

The aims of this study were to investigate descriptive parameters that may predict the solubility of compounds in the hydrofluoroalkane (HFA) propellants and to identify a model HFA propellant that is liquid at room temperature and atmospheric pressure. The solubility of 32 and 20 compounds chosen to give a wide range of physicochemical properties in HFA-134a and HFA-227, respectively, was measured. The Fedors solubility parameter and a computed log octanol water partition coefficient (CLOGP) were compared with the compounds' solubility in the HFA propellants. A total of 19 and 15 solutes had finite solubilities for HFA-134a and HFA-227, respectively, although the remaining solutes were miscible in all proportions. There was no apparent relation between solubility in HFA and the Fedors solubility parameter. This was not improved by considering the hydrogen-bonding potential of the compounds. When log solubility versus CLOGP was plotted, there was a linear relation for 16 and 12 of the compounds exhibiting a finite solubility in the HFA propellants, although four solutes (phenols) were displaced to the left of the linear relation. The remaining 3 compounds had much lower solubilities than was predicted from their CLOGPs, possibly as a consequence of their crystallinity (high melting points). Of the putative model propellants investigated (i.e., perfluorohexane (PFH), 1H-perfluorohexane [1H-PFH], and 2,2,2-trifluoroethanol), 1H-PFH was the most promising, with a linear relation between solubility in 1H-PFH and solubility in HFA propellant being observed. The solubilities in 1H-PFH were approximately 11 and 26% of those in HFA-134a and HFA-227.

Evaluation of the Accuracy and Precision of Lung Aerosol Deposition Measurements from Single-Photon Emission Computed Tomography Using Simulation—Fleming JS, Sauret V, Conway JH, Holgate ST, Bailey AG, Martonen TB. J Aerosol Med 2000;13(3):187-198.

Single-photon emission computed tomography (SPECT) imaging is being increasingly used to assess inhaled aerosol deposition. This study uses simulation to evaluate the errors involved in such measurements and to compare them with those from conventional planar imaging. SPECT images of known theoretical distributions of radioaerosol in the lung have been simulated using lung models derived from magnetic resonance studies in human subjects. Total lung activity was evaluated from the simulated images. A spherical transform of the lung distributions was performed, and the absolute penetration index (PI) and a relative value expressed as a fraction of that in a simulated ventilation image were calculated. All parameters were compared with the true value used in the

simulation, and the errors were assessed. An iterative method was used to correct for the partial volume effect, and its effectiveness in improving errors was evaluated. The errors were compared with those of planar imaging. The precision of measurements was significantly better for SPECT than planar imaging (2.8 vs 6.3% for total lung activity, 6 vs 20% for PI, and 3 vs 6% for relative PI). The method of correcting for the influence of the partial volume effect significantly improved the accuracy of PI evaluation without affecting precision. SPECT is capable of accurate and precise measurements of aerosol distribution in the lung, which are improved compared with those measured by conventional planar imaging. A technique for correcting the SPECT data for the influence of the partial volume effect has been described. Simulation is demonstrated as a valuable method of technique evaluation and comparison.

Respiratory-Related Quality of Life: Relation to Pulmonary Function, Functional Exercise Capacity, and Sputum Biophysical Properties—Piquette CA, Clarkson L, Okamoto K, Kim JS, Rubin BK. J Aerosol Med 2000;13(3):263-272.

One of the difficulties in assessing mucoactive therapy is selecting clinical outcome variables that reflect the impact of clearing airway secretions on quality of life (QOL). Petty and colleagues developed a questionnaire designed to evaluate the clinical impact of mucoactive therapy in patients with chronic bronchitis (CB). We evaluated this questionnaire in a multicenter study of a mucolytic medication used in patients with CB and hypothesized that spirometry, exercise capacity, and sputum clearability changes would correlate with QOL changes. This was a multicenter trial in 159 patients with stable CB (111 completed the 16-week study). Spirometry, plethysmography, the 6-minute walk test (6MWT), and Petty score as a measure of QOL were assessed at each visit. Sputum was collected at each visit. Cough transportability was measured in a cough machine, and mucociliary transportability was measured on the frog palate. Cohesivity was measured in a filancemeter, interfacial tension by de Nouy ring, and wettability by contact angle analysis. Within the entire data set of 694 evaluations, there was no correlation between pulmonary function and QOL. There was an inverse correlation with distance covered in a 6MWT ($R^2 = 0.041$, $p < 0.0001$). Sputum CTR was directly correlated with QOL ($R^2 = 0.027$, $p < 0.0001$). Change from baseline (mean of first three visits) was computed and compared the change in the mean of values at the 8- and 12-week visits (n = 108 sets of data pairs). This was analyzed as a percentage of change for continuous measurements, and as QOL is normative, we calculated the absolute change in QOL. There was no relation between QOL and 6MWT changes. There was an inverse relation between change in forced expiratory volume in 1 second and QOL ($R^2 = 0.092$, $p = 0.0021$) as well as between forced vital capacity and QOL ($R^2 = 0.05$, $p = 0.024$). There was a direct relation between CTR and QOL ($R^2 = 0.039$, $p = 0.048$). The relation between QOL and 6-minute walk distance was expected but weak. The consistent relation between CTR and QOL (suggesting that improved CTR of sputum is associated with decreased QOL) is difficult to explain. A change in forced expiratory volume in 1 second and forced vital capacity did correlate with a change in QOL. There is a need for a good QOL tool to evaluate mucus clearance devices or medications. The Petty questionnaire was designed specifically for this task, but the effect on sputum properties by current mucoactive agents may be too small to elicit a significant change in the Petty score.

Measuring Lung Function in Infancy—Lucas JS, Foreman CT, Clough JB. Respir Med 2000 Jul;94(7):641-647.

Although the earliest reliable lung function tests in infants were performed as long as 40 years ago, there has only recently been a growth in this area, as simpler methods and better equipment and IT resources have been developed. Exciting information is accumulating about the normal physiology and pathology of the infant lung. Many basic questions are

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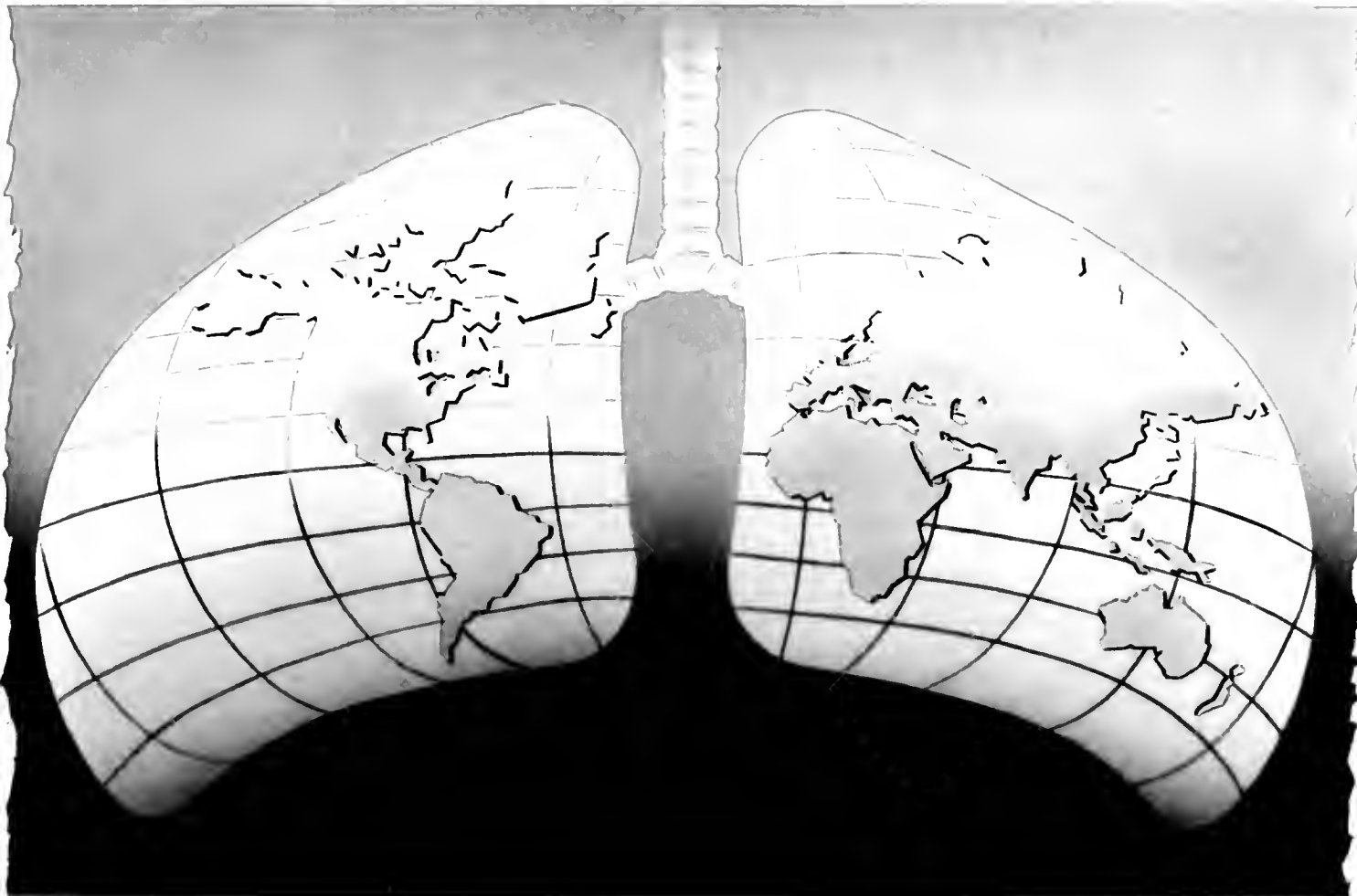
COMING UP FOR AIR

COPD is an umbrella term used to describe major lung diseases where airflow is reduced including emphysema and chronic bronchitis. The result is lots of coughing, shortness of breath, chest tightness and increased mucus production. And in severe cases, less oxygen and more carbon dioxide. Smoking is the culprit for 90 percent of all cases of COPD

Nearly half of the people diagnosed with COPD get short of breath while doing such mundane things as washing, dressing or doing light housework. A third get breathless just having a conversation or sitting or lying down.

The symptoms of COPD are globally under-recognized and sufferers of this condition tend to underestimate the severity of their symptoms. Do you have the symptoms?

COPD: Coming Up For Air, co-hosted by Stephen L. Rennard, M.D., Larson Professor of Medicine at the University of Nebraska Medical Center and James F. Donahue, M.D., Professor of Medicine at the University of North Carolina raises awareness of this under-reported illness.

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still unanswered and the ability to perform these tests remains confined to a few specialized centres. To co-ordinate the development of ILFT and establish standardization in a number of areas including measurement conditions, equipment specifications, methodology protocols and data analysis, international collaboration is necessary between the teams working in this field (Table 5). Collaborative groups are currently addressing these issues and are also developing recommendations regarding the design of randomized clinical trials, multi-centre studies and research agendas. Infant lung function testing remains primarily a research tool. Our aim should be not only to refine and develop the techniques of physiological measurement but to apply ILFT to the objective study of respiratory illness in infants in the clinical setting so as to aid in the prevention and treatment of these common, debilitating and costly diseases.

Comparison of High and Low Dose of the Inhaled Steroid, Budesonide, As an Initial Treatment in Newly Detected Asthma—Tukiainen H, Taivainen A, Majander R, Poussa T, Svahn T, Puolijoki H, Terho EO. Respir Med 2000 Jul;94(7):678-683

The importance of early initiation of inhaled steroids even in mild asthma has been documented in several studies. It is not, however, clear whether the treatment should be started with a high or a low dose of the inhaled steroid. We have compared the effects of high and low dose inhaled steroid, budesonide, in patients with newly detected asthma. We studied 101 adult patients with newly detected bronchial asthma who were without inhaled steroid or any regular pharmacological treatment for their asthma. The patients were randomly allocated to two treatment groups: one to receive 800 microg inhaled budesonide per day and the other to receive 200 microg inhaled budesonide per day. The drugs were given with a Turbuhaler dry powder inhaler. During the 3-month treatment period, no significant differences between the treatment groups were noted in morning or evening PEF values, in spirometric parameters, in asthmatic symptoms or in the use of rescue β_2-agonists. The decrease in bronchial hyperresponsiveness was, however, more marked in the high dose budesonide group, reaching a borderline significance (p = 0.10 high vs. low dose budesonide). In addition, in serum markers of asthmatic inflammation significant differences were shown between the treatment groups. The decrease in the number of blood eosinophils during the treatment was more marked in the high dose budesonide group (p = 0.02; high vs. low dose budesonide). In serum ECP no change was observed in the low dose budesonide group, but a marked decrease in the high-dose budesonide group (p = 0.008; high vs. low dose budesonide). The change was even more marked with regard to serum EPX (p = 0.005; high vs. low dose budesonide). Our results support the view that the treatment of newly detected asthma should be started with a high dose of inhaled steroid. The low dose may not be enough to suppress asthmatic inflammation despite good clinical primary response.

A Study to Investigate the Ability of Subjects with Chronic Lung Diseases to Provide Evidential Breath Samples Using the Lion Intoxilyzer 6000 UK Breath Alcohol Testing Device—Honeybourne D, Moore AJ, Butterfield AK, Azzan L. Respir Med 2000 Jul;94(7):684-688.

The Lion Intoximeter 3000 has been used for evidential breath testing in the UK for some years. Some individuals with lung diseases have difficulty in providing evidential breath samples using the device. This study describes an investigation that we have carried out on a newer instrument the Lion Intoxilyzer 6000UK which is now in use in the UK. The study was designed to investigate the ability of subjects with a variety of lung diseases to provide evidential breath samples using this device. The 40 adult subjects investigated comprised 10 normal controls, 10 with asthma, 10 with chronic obstructive pulmonary disease (COPD) and 10 with restrictive lung disease. After baseline spirometry, subjects

were given alcohol to drink, the quantity based upon body weight. After a gap of at least 20 min, subjects were asked to provide evidential breath samples in accordance with the test procedure built into the Lion Intoxilyzer 6000UK. The results showed that two asthmatic subjects, four with COPD and three with restrictive lung disease failed to provide evidential breath samples even after four attempts. Despite the device requiring a minimum sample volume of 1.2 L, eight of the nine subjects who failed had a forced vital capacity (FVC) of more than 1.5 L. Seven of these nine subjects had a forced expiratory volume in 1 sec (FEV_1) of less than 1.0 L. In conclusion, this study has shown that some subjects with lung diseases may have difficulty in providing evidential breath samples using the Lion Intoxilyzer 6000 UK.

Maximal Inspiratory Mouth Pressures (PIMAX) in Healthy Subjects: What Is the Lower Limit of Normal? Hautmann H, Hefele S, Schotten K, Huber RM. Respir Med 2000 Jul;94(7):689-693.

BACKGROUND: Maximal inspiratory mouth pressures are suitable for non-invasive evaluation of respiratory muscle function. Different studies on PIMAX give predicted normal values and their relation to anthropometric data. Due to a large inter-subject variation of PIMAX, predicted values, however, maximal inspiratory mouth pressures are not suitable to define the individual expected normal PIMAX. What is the lower limit of the normal range? METHODS: PIMAX has been prospectively measured in a representative sample of 504 healthy volunteers (248 males and 256 females) between 18 and 82 years of age with normal lung function. Age, height, weight, body mass index (BMI) and smoking status were recorded and incorporated stepwise in a multiple regression analysis to determine prediction equations. Lower limits of the normal range were defined as the fifth percentile of the residuals derived from the regression model. RESULTS: Mean values of PIMAX were 9.95 kPa for men and 7.43 kPa for women. Significant correlations were found with height, weight, BMI, FEV_1, PEF and FVC (p < 0.01). The strongest correlation appeared with sex and age (p < 0.001). Smoking status and smoked pack-years were not independent predictors of inspiratory pressures. Lower limits of normal were 59% for women and 60% for men of the predicted PIMAX. CONCLUSIONS: In the interpretation of maximal inspiratory mouth pressures, normal values should represent the lower limit of the normal range derived from the regression model in order to avoid false pathological results. Prediction equations as well as lower limits of normal resulting from a study cohort of healthy 18-82-year-olds are given and are recommended to be used by pulmonary function laboratories in young and old patients.

Airway Obstruction and Chronic Exertional Dyspnoea in Patients with Persistent Bronchial Asthma—Filippelli M, Pacini F, Romagnoli I, Rosi E, Ottanelli R, Duranti R, Scano G. Respir Med 2000 Jul;94(7):694-701.

In patients with COPD, flow limitation (FL) predicts chronic exertional dyspnoea (CED) better than routine spirometry. Whether, and to what extent, FL and CED are overlapping quantities in chronic asthma has not yet been defined. Forty consecutive clinically stable asthmatic patients without smoking history or cardiopulmonary disorders, were studied. In each subject respiratory function, including static and dynamic pulmonary volumes, was evaluated; maximal (MEFV) and partial (PEFV) expiratory V-V curves and isovolumic partial to maximal flow ratio (M/P). FL was assessed in a seated patient by comparing tidal and PEFV curves; FL was detected when tidal flows were superimposed or exceeded those obtained during PEFV curves, and was expressed as a percentage of the expired control tidal volume (V_T) affected by flow limitation ($FL\% V_T$). Dyspnoea was assessed by both MRC scale and Baseline Dyspnoea Index (BDI) focal score. Half of the patients were found to have FL. They were older, more dyspnoeic and more obstructed (p = 0.03 - p = 0.000005) than the non-FL group. FEV_1, vital capacity (VC), age, body mass index,

FL and M/P ratio were all related to dyspnoea scores. FL was significantly related to FEV_1 (r = - 0.59). Multiple regression analysis showed that FEV_1 (p=0.003, r^2 = 15.3% and p = 0.004, r^2 = 20.3%) and age (p = 0.0006, r^2 = 26.8% and p = 0.016, r^2 = 11%) independently predicted a part of the variance of MRC (p = 0.0001, r^2 = 42.1%) and BDI (p = 0.0008, r^2 = 31.3%), respectively. With dyspnoea scale being the gold standard, diagnostic accuracy (sensitivity and specificity) by ROC (receiver operating characteristics) analysis was similar for FEV_1 and FL. The results indicate that FL may be present in this subset of asthmatics. CED may not be easily explained by abnormalities of routine spirometry or FL, the largest part of the CED variance remained unexplained. Thus, routine spirometry, FL and CED in patients with bronchial asthma are only partially overlapping quantities which need to be assessed separately.

Inhaled Nebulized Adrenaline Improves Lung Function in Infants with Acute Bronchiolitis—Lodrup Carlsen KC, Carlsen KH. Respir Med 2000 Jul;94(7):709-714.

β_2-agonists have questionable symptomatic effect in infants with acute bronchiolitis, whereas inhaled, nebulized racemic adrenaline, commonly used in Norway, appears (clinically) to be effective. Limited lung function observations during acute bronchiolitis exists, and less for assessing possible effects inhaled adrenaline. In this preliminary study, tidal flow-volume loops were measured in 16 infants with acute bronchiolitis and seven healthy controls (mean age 7.9 and 4.4 months, respectively), with repeated measurements 15 min after inhaled nebulized racemic adrenaline (4 mg diluted in 2 mL saline) in nine bronchiolitis patients. The ratio of time to reach peak tidal expiratory flow to total expiratory time (tPTEF/tE) was significantly reduced in children with acute bronchiolitis (mean, 95% CI) (0.08, 0.05-0.10) compared to controls (0.31, 0.18-0.43), with significant improvement after inhaled racemic adrenaline 0.19 (0.13-0.25), parallel with significant clinical improvement. Lung function (tPTEF/tE) was reduced in infants with acute bronchiolitis and improved significantly after inhaled racemic adrenaline. Inhaled racemic adrenaline is potentially an important alternative for treating infants with acute bronchiolitis.

Effectiveness of a Clinical Pathway for Inpatient Asthma Management —Johnson KB, Blaisdell CJ, Walker A, Eggleston P. Pediatrics 2000 Nov;106(5):1006-1012.

BACKGROUND: Clinical pathways for asthma are tools that have the potential to improve compliance with nationally recognized management guidelines, but their effect on patient outcomes has not been documented. OBJECTIVES: To determine the effect of an asthma clinical pathway on patients' length of stay, use of nebulized beta-agonist therapy while hospitalized, and use of acute care clinics for 2 weeks after discharge. DESIGN/METHODS: The study was a randomized, controlled trial. Patients between the ages of 2 and 18 years admitted with an asthma exacerbation and not under the care of an asthma specialist were eligible for the study. Patients were randomized either to a conventional ward (control group) or to a ward using the clinical pathway (intervention group). For 2 weeks after discharge, we collected data to determine whether patients visited a health care provider for worsening asthma. RESULTS: One hundred ten patients (26%) were enrolled. Control and intervention groups had similar demographic and asthma severity profiles. The intervention group had an average length of stay 13 hours shorter than did the control group. In addition, at every dosing interval, the intervention group received less nebulized beta-agonist therapy. There were no deaths in either group. CONCLUSION: A clinical pathway for inpatient asthma decreased the length of stay and beta-agonist medication use with no adverse outcomes or increased acute-care encounters through 2 weeks after discharge.

Respiratory Symptoms in Mothers of Young Children—d'Arcy H. Gillespie B. Foxman B. Pediatrics 2000 Nov;106(5):1013-1016.

OBJECTIVES: Children receiving child care outside the home are at greater risk of upper respiratory infection, but whether parents of those children are also at increased risk is undocumented. We describe the incidence of 2 or more respiratory symptoms in the previous 2 weeks among 185 mothers of children 3 years of age or younger by child care use. METHODS: Mothers in Michigan and Nebraska were interviewed by phone regarding respiratory symptoms, use of outside child care (for an index child), sleeping habits, and demographic information. RESULTS: Nearly one half (46.5%) reported 2 or more symptoms during the past 2 weeks; 15.1% had contacted a health care provider and 13.0% spent 1 or more days in bed because of their symptoms, which lasted an average of 5.5 days. Prevalence of symptoms was invariant to sociodemographic characteristics. Mothers using outside child care (74.6%) were twice as likely as those without outside care to have been ill in the past 2 weeks (odds ratio: 2.26, 95% confidence interval [CI]: 1.12,4.54). Most mothers (69.2%) reported having their sleep interrupted by their children at least once in the last 2 weeks or sharing a bed with a child part or all of the night (61.1%); 25.4% slept 6 hours or less nightly. Women reporting that they rarely or never felt rested (26. 5%) were 2.65 times more likely to be ill (95% CI: 1.26,5.55), compared with those reporting that they frequently or always felt rested (46.5%), after adjusting for any outside child care. CONCLUSIONS: Future studies should focus on risk factors that can be modified to reduce illness among both children and their parents.

Can Epinephrine Inhalations Be Substituted for Epinephrine Injection in Children at Risk for Systemic Anaphylaxis?—Simons FE, Gu X, Johnston LM, Simons KJ. Pediatrics 2000 Nov;106(5):1040-1044

BACKGROUND: For out-of-hospital treatment of anaphylaxis, inhalation of epinephrine from a pressurized metered-dose inhaler is sometimes recommended as a noninvasive, user-friendly alternative to an epinephrine injection. OBJECTIVE: To determine the feasibility of administering an adequate epinephrine dose from a metered-dose inhaler in children at risk for anaphylaxis by assessing the rate and extent of epinephrine absorption after inhalation. METHODS: We performed a prospective, randomized, observer-blind, placebo-controlled, parallel-group study in 19 asymptomatic children with a history of anaphylaxis. Based on the child's weight, 10, 15, or 20 carefully supervised epinephrine or placebo inhalations were attempted. Before dosing, and at intervals from 5 to 180 minutes after dosing, we monitored plasma epinephrine concentrations, blood glucose, heart rate, blood pressure, and adverse effects. RESULTS: Eleven children (mean ± standard error of the mean: 9 ± 1 years and 33 ± 3 kg) in the epinephrine group were able to inhale 11 ± 2 (range: 3-20) putts, equivalent to 74% ± 7% of the precalculated dose or 0.078 ± 0.009 mg/kg. They achieved a mean peak plasma epinephrine concentration of 1822 ± 413 (range: 230-4518) pg/mL at 32.7 ± 6.2 minutes. Eight children (10 ± 1 years of age and 33 ± 5 kg) in the placebo group were able to inhale 12 ± 2 (range: 8-20) putts, 89% ± 3% of the precalculated dose, and had a peak endogenous plasma epinephrine concentration of 1316 + 247 (range: 522-2687) pg/mL at 44.4 ± 16.7 minutes. In the children receiving epinephrine compared with those receiving placebo, mean plasma epinephrine concentrations were not significantly higher at any time, mean blood glucose concentrations were significantly higher from 10 to 30 minutes, mean heart rate was not significantly different at any time, and mean systolic and diastolic blood pressures were not significantly increased at most times. After the inhalations of epinephrine or placebo, the children complained of bad taste and many experienced cough or dizziness. After inhaling epinephrine, 1 child developed nausea, pallor, and muscle twitching. CONCLUSIONS: Despite expert coaching, because of the number of epinephrine inhalations required and the bad taste of the inhalations, most children were

unable to inhale sufficient epinephrine to increase their plasma epinephrine concentrations promptly and significantly. Therefore, we urge caution in recommending epinephrine inhalation as a substitute for epinephrine injection for out-of-hospital treatment of anaphylaxis symptoms in children.

Forgoing Life-Sustaining Medical Treatment in Abused Children. Pediatrics 2000 Nov;106(5):1151-1153.

A decision to forgo life-sustaining medical treatment (LSMT) for a critically ill child injured as the result of abuse should be made using the same criteria as those used for any critically ill child. The parent or guardian of an abused child may have a conflict of interest when a decision to forgo LSMT risks changing the legal charge faced by a parent, guardian, relative, or acquaintance from assault to manslaughter or homicide. If a physician suspects that a parent or guardian is not acting in a child's best interest, further review and consultation should be sought in hopes of resolving the conflict. A guardian ad litem who will represent the child's interests regarding LSMT should be appointed in all cases in which a parent or guardian may have a conflict of interest.

Effects of Nasal Continuous Positive Airway Pressure on Soluble Cell Adhesion Molecules in Patients with Obstructive Sleep Apnea Syndrome—Chin K, Nakamura T, Shimizu K, Mishima M, Nakamura T, Miyasaka M, Ohi M. Am J Med 2000 Nov;109(7):562-5657.

PURPOSE: Obstructive sleep apnea syndrome is common in middle-aged men and may be associated with an increased risk of cardiovascular disease. We investigated the effect of nasal continuous positive airway pressure (CPAP) treatment on levels of soluble cell adhesion molecules-which have been shown to be associated with the development of atherosclerosis-in these patients. SUBJECTS AND METHODS: We studied 23 patients with obstructive sleep apnea syndrome diagnosed by polysomnography who were treated with nasal CPAP. Serum soluble intercellular adhesion molecule-1, E-selectin, and vascular cell adhesion molecule-1 levels were measured before nasal CPAP was started, and after 3 or 4 days (n = 19), 1 month (n = 23), or 6 months (n = 11) of treatment. RESULTS: After 3 to 4 days of nasal CPAP therapy, the mean (± SD) soluble E-selectin level had decreased from 89 ± 44 ng/mL to 69 ± 28 ng/mL (p = 0.002). After 1 month, the soluble intercellular adhesion molecule-1 level had decreased from 311 ± 116 ng/mL to 249 ± 74 ng/mL (p = 0.02). After 6 months, soluble vascular cell adhesion molecule-1 levels had not changed significantly, while the mean soluble intercellular adhesion molecule-1 level (212 ± 59 ng/mL) had decreased further (p = 0.02). Before treatment, soluble intercellular adhesion molecule-1 levels and the apnea and hypopnea index were correlated (r = 0.43, p = 0.04) CONCLUSIONS: Obstructive sleep apnea and hypopnea have a significant adverse effect on serum soluble cell adhesion molecule-1 levels that may be reduced by nasal CPAP treatment.

Evaluation of a New Module in the Continuous Monitoring of Respiratory Mechanics—Nunes S, Takala J. Intensive Care Med 2000 Jun;26(6):670-678

OBJECTIVE: Bedside monitoring of respiratory mechanics facilitates the use of lung protective ventilation in acute lung injury (ALI). We evaluated a new clinical monitor of respiratory mechanics. DESIGN: Prospective, in vitro and in vivo study. SETTING: University hospital. PATIENTS: Measurements were done using a lung model and in patients after cardiac surgery (n = 10) and in patients with ALI (n = 10). INTERVENTIONS AND MEASUREMENTS: The monitor provides continuous monitoring of pressure, flow and volume waveform and loop data, and automatically collected variables of respiratory mechanics. Breath-by-breath respiratory mechanics data and the automated variables obtained with the new monitor were compared with flow and pressure

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reference data. RESULTS: Waveform data comparison showed errors of less than 5% for most variables. Automatically recorded respiratory pressures and volumes showed good agreement within clinical standards when compared to reference (errors from 2.5% to 6.2%). Automatically recorded derived variables present poor agreement (errors from 8.1% to 158.3%). CONCLUSIONS: The waveform data of the new monitor is accurate. The value of the automatically derived variables is limited by the fact that inspiratory plateau pressure and plateau compliance have no direct physiological meaning. Nevertheless, in clinical monitoring much information can be derived from the waveform signals alone and from pressure-volume and flow-volume loops. These facilitate monitoring changes in respiratory mechanics in the ALI patient.

The Effect of Lung Injury and Excessive Lung Fluid, on Impedance Cardiac Output Measurements, in the Critically Ill—Critchley LA, Calcroft RM, Tan PY, Kew J, Critchley JA. Intensive Care Med 2000 Jun;26(6):679-685.

OBJECTIVES: To investigate the relationship between the attenuation of impedance cardiac output (IC_{co}) measurements and lung fluid content in critically ill patients. DESIGN: Observational study. SETTING: Intensive Care Unit of a major teaching hospital in Hong Kong. PATIENTS: Twenty-four critically ill patients who required a pulmonary artery catheter. MEASUREMENTS AND MAIN RESULTS: Triplicate thermodilution cardiac output (TD_{co}) and BoMed NCCOM3 (IC_{co}) measurements were made simultaneously on a single occasion in each patient. Lung fluid accumulation was assessed by: (a) thoracic impedance (Zo), (b) radiological assessment of chest x-rays using an alveolar consolidation score (0-4) and (c) scoring the degree of hypoxia and use of positive end-expiratory pressure (PEEP). Offsets (TD_{co}-IC_{co})/TD_{co}, expressed as percentage, were compared with these indices of excess lung fluid. Patients were divided into those with sepsis (n = 13), fluid balance problems (n = 5) and cardiothoracic problems (n = 6). Mean cardiac output values were: 6.7 L/min TD_{co} (range 3.6-12.9) and 5.2 L/min IC_{co} (range 2.7-9.0). Overall the TD_{co} and IC_{co} values showed great variance, with a bias and limits of agreement of 1.49 ± 4.16 L/min, or ± 69%. In septic patients, increasing offset was correlated with decreases in Zo (r = 0.73, p = 0.005) and increases in alveolar consolidation score (r = 0.72, p = 0.005). CONCLUSIONS: The BoMed under-estimates cardiac output in critically ill patients. In septic patients the degree of attenuation of IC_{co} can be related to the extent of lung injury and fluid accumulation within the thorax.

Failure of a Brief Educational Program to Improve Interpretation of Pulmonary Artery Occlusion Pressure Tracings—Zarich S, Pust-Marcone J, Amoateng-Adjepong Y, Manthous CA. Intensive Care Med 2000 Jun;26(6):698-703.

OBJECTIVE: To determine whether a brief educational program can reduce variability of interpretation of pulmonary artery occlusion pressure (PAOP) tracings. DESIGN: Prospective, observational study. PARTICIPANTS: Twenty-three intensive care nurses and 18 physicians. INTERVENTIONS: Participants interpreted PAOP tracings before and 1 week after receiving a single, brief educational session and/or written materials ("in-service") designed to reduce interobserver variability of PAOP interpretation. Differences between two reference values before and after in-service (mean population and Chief of Critical Care's readings) were compared for both groups. RESULTS: There were no significant differences in the variabilities in PAOP interpretations before and after in-service in either group. CONCLUSIONS: We conclude that this specific educational program was ineffective in reducing variability of interpretation of PAOP tracings. These data suggest that more comprehensive educational tools and/or sustained programs may be required to improve performance of critical care personnel in PAOP interpretation.

Comparison of a Specialist Retrieval Team with Current United Kingdom Practice for the Transport of Critically Ill Patients—Bellingam G, Olivier T, Batson S, Webb A. Intensive Care Med 2000 Jun;26(6):740-744.

OBJECTIVE: The inter-hospital transfer of critically ill patients in the United Kingdom is commonly undertaken using standard ambulance under junior doctor escort, despite recommendations for the use of specialist retrieval teams. Patients are transferred into University College London Hospitals (UCLH) intensive care unit (ICU) by both methods. We undertook to evaluate the effect of transfer method on acute physiology (within 2 h of ICU admission) and early mortality (< 12 h after ICU admission). DESIGN: Retrospective review of all transfers over 1 year. SETTING: UCLH ICU. SUBJECTS: 259 transfers; 168 by specialist retrieval team (group A) and 91 by standard ambulance with doctor provided by referring hospital (group B). INTERVENTIONS: None. MAIN OUTCOME MEASURES: Acute physiology (pH, P_{aO_2}, P_{aCO_2}, heart rate (HR), mean arterial blood pressure (MAP), 24 h severity of illness scores (APACHE II, SAPS II), length of stay and mortality. RESULTS: There were no differences in demographic characteristics or severity of illness between the two groups; nevertheless significantly more patients in group B than in group A were severely acidotic (pH < 7.1: 11% vs. 3%, p < 0.008) and hypotensive (MAP < 60: 18 % vs. 9%, p < 0.03) upon arrival. In addition, there were more deaths within the first 12 h after admission with 7.7 % deaths (7/91) in group B transfers vs. 3% (5/168) in group A. CONCLUSIONS: The use of a specialist transfer team may significantly improve the acute physiology of critically ill patients and may reduce early mortality in ICU.

Lung Recruitment and Lung Volume Maintenance: A Strategy for Improving Oxygenation and Preventing Lung Injury During Both Conventional Mechanical Ventilation and High-Frequency Oscillation—Rimensberger PC, Pache JC, McKerlie C, Frndova H, Cox PN. Intensive Care Med 2000 Jun;26(6):745-755.

OBJECTIVE: To determine whether using a small tidal volume (5 mL/kg) ventilation following sustained inflation with positive endexpiratory pressure (PEEP) set above the critical closing pressure (CCP) allows oxygenation equally well and induces as little lung damage as high-frequency oscillation following sustained inflation with a continuous distending pressure (CDP) slightly above the CCP of the lung. MATERIAL AND METHODS: Twelve surfactant-depleted adult New Zealand rabbits were ventilated for 4 h after being randomly assigned to one of two groups: group 1, conventional mechanical ventilation, tidal volume 5 mL/kg, sustained inflation followed by PEEP > CCP; group 2, high-frequency oscillation, sustained inflation followed by CDP > CCP. RESULTS: In both groups oxygenation improved substantially after sustained inflation (p < 0.05) and remained stable over 4 h of ventilation without any differences between the groups. Histologically, both groups showed only little airway injury to bronchioles, alveolar ducts, and alveolar airspace, with no difference between the two groups. Myeloperoxidase content in homogenized lung tissue, as a marker of leukocyte infiltration, was equivalent in the two groups. CONCLUSIONS: We conclude that a volume recruitment strategy during small tidal volume ventilation and maintaining lung volumes above lung closing is as protective as that of high-frequency oscillation at similar lung volumes in this model of lung injury.

Preliminary Results on Nursing Workload in a Dedicated Weaning Center—Vitacca M, Clini E, Porta R, Ambrosino N. Intensive Care Med 2000 Jun;26(6):796-799.

OBJECTIVE: To evaluate the nursing time required for difficult-to-wean patients in a dedicated weaning center (WC) and to examine the correlation of the nursing time with nursing workload (NW) scores and with

clinical severity and dependency. SETTING: Ten-bed WC of a pulmonary rehabilitation department. INTERVENTION: None. DESIGN AND MEASUREMENT: Prospective, observational study of 46 consecutive patients admitted to a long-term WC. Time required by items of the Time Oriented Score System (TOSS) and other tasks specific to respiratory intermediate intensive care units were evaluated for all the activities performed on each patient in the first 2 days after admission. Patient dependency and level of nursing care at admission were measured using the Dependence Nursing Scale (DNS) and the Intermediate Therapeutic Intervention Score System (TISS int). The Acute Physiology and Chronic Health Evaluation (APACHE II) score was also recorded at admission. RESULTS: On the first day each patient needed $45 \pm 15\%$, $163 \pm 23\%$, $45 \pm 22\%$, and $29 \pm 11\%$ for the three nursing shifts of allocated single nursing time. On the TOSS on the first day patients required a daily mean $28 \pm 10\%$ of total available nursing time, on the second day the results did not change. Time of care in the first 24 h was only weakly related to DNS, APACHE II score, and TISS int; only DNS was able (although weakly, $r = 0.45$) to predict minutes of nursing care. CONCLUSIONS: In difficult-to-wean patients from mechanical ventilation the nursing time in the first 2 days after admission is high. The use of TOSS may underestimate NW by about 38%. Although only DNS showed the ability to predict minutes of care, the weak relationship limits its value in clinical practice.

Midazolam and 2% Propofol in Long-Term Sedation of Traumatized Critically Ill Patients: Efficacy and Safety Comparison—Sandrumenge Camps A, Sanchez-Izquierdo Riera JA, Toral Vazquez D, Sa Borges M, Peinado Rodriguez J, Alted Lopez E. Crit Care Med 2000 Nov;28(11):3612-3619.

OBJECTIVE: We proposed to compare the efficacy and safety of midazolam and propofol in its new preparation (2% propofol) when used for prolonged, deep sedation in traumatized, critically ill patients. We retrospectively compared 2% propofol with its original preparation, 1% propofol, used in a previous study in a similar and contemporary set of patients. DESIGN: A prospective, randomized, unblinded trial (midazolam and 2% propofol) and a retrospective, contemporary trial (2% propofol and 1% propofol). SETTINGS: A trauma intensive care unit in a tertiary university hospital. PATIENTS: A total of 63 consecutive trauma patients, admitted within a period of 5 months and requiring mechanical ventilatory support for > 48 hrs, 43 of whom (73%) suffered severe head trauma. We also retrospectively compared the 2% propofol group with a series of patients in whom 1% propofol was used. INTERVENTIONS: For the prospective trial, we randomized two groups: a midazolam group with continuous administration of midazolam at dosages 0.1-0.35 mg/kg/hr, and a 2% propofol group with continuous infusion at dosages 1.5-6 mg/kg/hr. Equal dosages of analgesics were administered. Similar management protocols were applied in the 1% propofol group used in the retrospective analysis with 2% propofol. MEASUREMENTS AND MAIN RESULTS: Epidemiologic and efficacy variables were recorded. Hemodynamic and biochemical variables were also monitored on a regular basis. Neuromonitoring was also performed on those patients with head trauma. Sedation adequacy was similar and patient behavior after drug discontinuation was not different in either prospective group (midazolam and 2% propofol). Hemodynamic or neuromonitoring variables were also similar for both groups. Triglyceride levels were significantly higher in the 2% propofol group compared with the midazolam group. A higher number of therapeutic failures because of sedative inefficacy was seen in the 2% propofol group compared with the midazolam group, especially during the first sedation days. When comparing 2% propofol and 1% propofol, a significantly higher number of therapeutic failures because of hypertriglyceridemia were found in the 1% propofol group, as opposed to a major number of therapeutic failures because of inefficacy found in the

2% propofol group. CONCLUSIONS: Propofol's new preparation is safe when used in severely traumatized patients. Its more concentrated formula improves the lipid overload problem seen with the prolonged use of the previous preparation. Nevertheless, a major number of therapeutic failures were detected with 2% propofol because of the need for dosage increase. This fact could be caused by a different disposition and tissue distribution pattern of both propofol preparations. New studies will be needed to confirm these results.

Effectiveness of End-Tidal Carbon Dioxide Tension for Monitoring Thrombolytic Therapy in Acute Pulmonary Embolism —Wiegand UK, Kurowski V, Giannitsis E, Katus HA, Djonlagic H. Crit Care Med 2000 Nov;28(11):3588-3592

OBJECTIVE: In acute massive pulmonary embolism with hemodynamic instability, monitoring of pulmonary artery pressure can be used to assess the efficacy of thrombolytic therapy. As a noninvasive alternative to pulmonary artery catheterization, we investigated the efficacy of continuous monitoring of end-tidal CO_2 tension. DESIGN: In 12 patients with massive pulmonary embolism who required mechanical ventilation, mean pulmonary arterial pressure (MPAP) and end-tidal carbon dioxide tension (ET_{CO_2}) were registered continuously during thrombolytic therapy. P_{aCO_2}, cardiac index as estimated by thermodilution catheter and respiratory ratio of arterial oxygen tension and inhaled oxygen concentration (P_{aO_2}/F_{IO_2}) were determined every 60 mins. MEASUREMENTS AND MAIN RESULTS: Before thrombolysis, MPAP (34.5 ± 9.8 mm Hg) and the difference between P_{aCO_2} and ET_{CO_2} (10.1 ± 4.7 mm Hg) were markedly increased compared with normal values. Continuously monitored MPAP was related to ET_{CO_2} for both all patients ($r^2 = 0.42$, p < 0.001) and individually (mean $r^2 = 0.92$, range, 0.79-0.98, p < 0.001). In ten survivors, the mean cardiac index and P_{aO_2}/F_{IO_2} increased during therapy from 1.7 ± 0.4 to 2.8 ± 0.6 L/min × m² and 125 ± 27 to 285 ± 50 mm Hg (p < 0.01, respectively). In these patients, the difference between P_{aCO_2} and ET_{CO_2} decreased from 9.8 ± 4.5 to 2.8 ± 0.9 mm Hg (p < 0.001). Recurrent embolism was detected in two patients by sudden reduction of ET_{CO_2}. CONCLUSIONS: Analysis of ET_{CO_2} allows monitoring of the efficacy of thrombolysis and may reflect recurrent embolism. Thus, on the basis of this small study, analysis of ET_{CO_2} appears to be useful for noninvasive monitoring in mechanically ventilated patients with massive pulmonary embolism.

Effects of Inspired Gas Content During Respiratory Arrest and Cardiopulmonary Resuscitation—Idris AH. Crit Care Med 2000 Nov;28(11 Suppl):N196-N198.

Mouth-to-mouth and bag-valve-mask ventilation have been an indispensable part of cardiopulmonary resuscitation (CPR). However, only recently have the effects of different tidal volumes on arterial oxygenation been reported for mouth-to-mouth and bag-valve-mask ventilation. Currently recommended tidal volumes (10-15 mL/kg) are associated with an increased risk of gastric inflation because they produce high peak inspiratory pressures. An animal model of ventilation with an unprotected airway showed that a smaller tidal volume (6 mL/kg) is as effective as a larger tidal volume (12 mL/kg) in maintaining S_{aO_2} at ~96%. However, a smaller tidal volume with exhaled gas ventilation produced a mean S_{aO_2} of 48%, which is ineffective. Ventilation gas mixtures have been studied in models of cardiac arrest and CPR. One study showed that ventilation with air during 6 mins of CPR resulted in a return of spontaneous circulation in 10 of 12 animals compared with only 5 of 12 animals ventilated with exhaled gas (p < 0.04). Arterial and mixed-venous P_{O_2} were significantly higher, and P_{CO_2} was significantly lower in the air ventilation group. Investigations of the cardiovascular effects of mouth-to-mouth ventilation during CPR suggest that there are adverse effects during low blood flow states. However, mouth-to-mouth ventilation during respiratory arrest is lifesaving and should continue to be taught and emphasized in basic life support courses.

Percutaneous Tracheostomy in Critically Ill Patients: A Prospective, Randomized Comparison of Two Techniques—Nates JL, Cooper DJ, Myles PS, Scheinkestel CD, Tuxen DV. Crit Care Med 2000 Nov;28(11):3734-3739.

OBJECTIVE: To prospectively compare two commonly used methods for percutaneous dilational tracheostomy (PDT) in critically ill patients. DESIGN: Prospective, randomized, clinical trial. SETTING: Trauma and general intensive care units of a university tertiary teaching hospital, which is also a level 1 trauma center. PATIENTS: One hundred critically ill patients with an indication for PDT. INTERVENTIONS: PDT with the Ciaglia technique using the Ciaglia PDT introducer set and the Griggs technique using a Griggs PDT kit and guidewire dilating forceps. MEASUREMENTS AND MAIN RESULTS: Surgical time, difficulties, and surgical and anesthesia complications were measured at 0-2 hrs, 24 hrs, and 7 days postprocedure. Groups were well matched, and there were no differences between the two methods in surgical time or in anesthesia complications. Major bleeding complications were 4.4 times more frequent with the Griggs PDT kit. With the Ciaglia PDT kit, both intraoperative and at 2 and 24 hrs, surgical complications were less common (p = 0.023) and the procedure was more often completed without expert assistance (p = 0.013). Tracheostomy bleeding was not associated with either anticoagulant therapy or an abnormal clotting profile. Multivariate analysis identified the predictors of PDT complications as the Griggs PDT kit (p = 0.027) and the Acute Physiology and Chronic Health Evaluation (APACHE) II score (p = 0.041). The significant predictors of time required to complete PDT were the APACHE II score (p = 0.041), a less experienced operator (p = 0.0001), and a female patient (p = 0.013). CONCLUSIONS: Patients experiencing PDT with the Ciaglia PDT kit had a lower surgical complication rate (2% vs. 25%), less operative and postoperative bleeding, and less overall technical difficulties than did patients undergoing PDT with the Griggs PDT kit. Ciaglia PDT is, therefore, the preferred technique for percutaneous tracheostomy in critically ill patients.

Comparison of the Response of Saline Tonometry and an Automated Gas Tonometry Device to a Change in CO_2—Noone RB, Bolden JE, Mythen MG, Vaslef SN. Crit Care Med 2000 Nov;28(11):3728-3733.

OBJECTIVE: To examine the speed of response of saline tonometry and an automated gas tonometry system by using standard tonometry catheters. DESIGN: In vitro validation study. SETTING: Experimental research laboratory. INTERVENTIONS: Tonometry catheters were placed in a test chamber designed to simulate the lumen of a hollow viscus and were exposed to a rapid change in CO_2 from 0% to 5% or 10%. Measured CO_2 over time was fit to a mathematical model to determine the response time constant (the time to reach 63% of the final value) for each system. MEASUREMENTS AND MAIN RESULTS: Response time to a change in CO_2 was significantly faster with the automated gas system than with traditional saline tonometry. The mathematical time constant for a 5% change in CO_2 in a gas environment was 2.8 mins (95% confidence interval, 2.6-3.0 mins) for the gas and 6.3 mins (95% confidence interval, 5.8-7.3 mins) for the saline technique. These times were longer for the CO_2 change in a liquid environment. The time constant was 4.6 mins (95% confidence interval, 4.5-4.7 mins) for the gas system and 7.8 mins (95% confidence interval, 7.15-8.6 mins) for the saline tonometry. There was a significantly lower final equilibration value for the CO_2 measurement with saline tonometry. There was essentially no difference in time constants for each system for a 5% change compared with a 10% CO_2 change, except for a slightly faster time constant for the gas tonometry system with a 5% change in the gas environment (5%: 2.8 mins vs. 10%: 3.3 mins). CONCLUSIONS: The automated gas tonometry system has a

significantly faster response to a change in CO₂ than conventional saline tonometry

Inhaled Nitric Oxide Reduces the Need for Extracorporeal Membrane Oxygenation in Infants with Persistent Pulmonary Hypertension of the Newborn
Christou H, Van Marter LJ, Wessel DL, Allred EN, Kane JW, Thompson JE, et al. Crit Care Med 2000 Nov;28(11): 3722-3727

OBJECTIVE: We previously reported improved oxygenation, but no change, in rates of extracorporeal membrane oxygenation (ECMO) use or death among infants with persistent pulmonary hypertension of the newborn who received inhaled nitric oxide (NO) with conventional ventilation, irrespective of lung disease. The goal of our study was to determine whether treatment with inhaled NO improves oxygenation and clinical outcomes in infants with persistent pulmonary hypertension of the newborn and associated lung disease who are ventilated with high-frequency oscillatory ventilation (HFOV). DESIGN: Single-center, prospective, randomized, controlled trial. SETTING: Newborn intensive care unit of a tertiary care teaching hospital. PATIENTS: We studied infants with a gestational age of >34 wks who were receiving mechanical ventilatory support and had echocardiographic and clinical evidence of pulmonary hypertension and hypoxemia (P$_{aO_2}$ < 100 mm Hg on F$_{IO_2}$ = 1.0), despite optimal medical management. Infants with congenital heart disease, diaphragmatic hernia, or other major anomalies were excluded. INTERVENTIONS: The treatment group received inhaled NO, whereas the control group did not. Adjunct therapies and ECMO criteria were the same in the two groups of patients. Investigators and clinicians were not masked as to treatment assignment, and no crossover of patients was permitted. MEASUREMENTS AND MAIN RESULTS: Primary outcome variables were mortality and use of ECMO. Secondary outcomes included change in oxygenation and duration of mechanical ventilatory support and supplemental oxygen therapy. Forty-two patients were enrolled. Baseline oxygenation and clinical characteristics were similar in the two groups of patients. Infants in the inhaled NO group (n = 21) had improved measures of oxygenation at 15 mins and 1 hr after enrollment compared with infants in the control group (n = 20). Fewer infants in the inhaled NO group compared with the control group were treated with ECMO (14% vs. 55%, respectively, p = 0.007). Mortality did not differ with treatment assignment. CONCLUSIONS: Among infants ventilated by HFOV, those receiving inhaled NO had a reduced need for ECMO. We speculate that HFOV enhances the effectiveness of inhaled NO treatment in infants with persistent pulmonary hypertension of the newborn and associated lung disease.

Improved Outcomes of Children with Malignancy Admitted to a Pediatric Intensive Care Unit
Hallahan AR, Shaw PJ, Rowell G, O'Connell A, Schell D, Gillis J. Crit Care Med 2000 Nov;28(11):3718-3721.

OBJECTIVE: To assess the acute and long-term outcomes of children admitted to the intensive care unit with cancer or complications after bone marrow transplantation. DESIGN: Retrospective analysis of databases from a prospective pediatric intensive care unit (PICU) database supplemented by case notes review. SETTING: A PICU in a tertiary pediatric hospital. PATIENTS: All children with malignancy admitted to the PICU between May 1, 1987, and April 30, 1996. INTERVENTIONS: None. MEASUREMENTS AND MAIN RESULTS: There were 206 admissions to the PICU during a 9-yr study period of 150 children with malignancies or complications after bone marrow transplantation. Forty patients died in the PICU (27% mortality rate). The most frequent indications for PICU admission were shock and respiratory disease. Of 56 children admitted with shock, there were 16 deaths (29% mortality rate). In 24 episodes of sepsis, inotropic and ventilatory support were required and 13 patients (54%) survived. Analysis of long-term survival gave

estimates of 50% survival for all oncology patients admitted to the PICU and 42% for those admitted for shock. CONCLUSIONS: A high proportion of oncology patients admitted to the PICU requiring intensive intervention survive and go on to be cured of their malignancy. Our study suggests the PICU outcome for these patients has improved.

An Improved In Vivo Rat Model for the Study of Mechanical Ventilatory Support Effects on Organs Distal to the Lung
Valenza F, Sibilla S, Porro GA, Brambilla A, Tredici S, Nicolini G, et al. Crit Care Med 2000 Nov;28(11):3697-3704

OBJECTIVE: To study the influence of different mechanical ventilatory support strategies on organs distal to the lung, we developed an in vivo rat model, in which the effects of different tidal volume values can be studied while maintaining other indexes. DESIGN: Prospective, randomized animal laboratory investigation. SETTING: University laboratory of Ospedale Maggiore di Milano-Instituto di Ricovero e Cura a Carattere Scientifico. SUBJECTS: Anesthetized, paralyzed, and mechanically ventilated male Sprague-Dawley rats. INTERVENTIONS: Two groups of seven rats each were randomized to receive tidal volumes of either 25% or 75% of inspiratory capacity (IC), calculated from a preliminary estimation of total lung capacity. Ventilation strategies for the two groups were as follows: a) 25% IC, 9.9 ± 0.8 mL/kg, frequency, 59 ± 4 beats/min; positive end-expiratory pressure, 3.6 ± 0.8 cm H₂O, and peak inspiratory airway pressure (P$_{aw}$), 13.2 ± 2 cm H₂O, and b) 75% IC, 29.8 ± 2.9, frequency, 23 ± 13; positive end-expiratory pressure, 0; peak inspiratory P$_{aw}$, 29.0 ± 3. MEASUREMENTS AND MAIN RESULTS: Mean arterial pressure (invasively monitored) remained well above adequate perfusion pressure values throughout, and no significant difference was seen between the two groups. P$_{aO_2}$, pHa, and P$_{aCO_2}$ values were compared after 60 mins of ventilation and again, no significant difference was seen between the two groups (P$_{aO_2}$, 269 ± 25 and 260 ± 55 torr, pHa, 7.432 ± 0.09 and 7.415 ± 0.03, P$_{aCO_2}$, 35.4 ± 8 and 32.5 ± 2 torr, for the 25% IC and 75% IC groups, respectively). Mean P$_{aw}$ were not different (6.4 ± 0.8 cm H₂O in the 25% IC groups, and 6.1 ± 1.2 in the 75% IC groups, respectively). At the end of the experiment, animals were killed and the liver and kidney isolated, fixed in 4% formalin, cut, and stained for optic microscopy. Kidneys from rats ventilated with 75% IC showed increased Bowman's space with collapse of the glomerular capillaries. This occurred in a greater percentage of rats ventilated with 75% IC (0.67 ± 0.2 vs. 0.29 ± 0.2, 75% IC vs. 25% IC, respectively, p < 0.05). Perivascular edema was also present in rats ventilated with 75% IC (p < 0.05). Morphometric determinations of the empty zones (index of edema) demonstrated a trend toward differences between 75% IC livers and 25% IC (0.14 ± 0.05 vs. 0.11 ± 0.02, respectively). CONCLUSION: We conclude that it is possible to study the effects of mechanical ventilatory support on organs distal to the lung by means of an in vivo rat model.

The Comfort of Breathing: A Study with Volunteers Assessing the Influence of Various Modes of Assisted Ventilation
Russell WC, Greer JR. Crit Care Med 2000 Nov;28(11):3645-3648.

OBJECTIVE: To assess the subjective feeling of comfort of healthy volunteers breathing on various modes of ventilation used in intensive care. DESIGN: A randomized, prospective, double-blinded, crossover trial using volunteers. SETTING: An intensive care unit (ICU) in a teaching hospital. INTERVENTIONS: We compared, by using healthy volunteers, the subjective feeling of comfort of three modes of ventilation used during the weaning phase of critical illness. We used healthy volunteers to avoid other distracting influences of intensive care that may confound the primary feeling of comfort. The modes we compared were synchronized intermittent mandatory ventilation, assisted spontaneous breathing, and biphasic positive airway pressure. The imposed ventilation was comparable with 50% of the volunteers' normal respiratory effort. The volunteers breathed via a mouthpiece through a ventilator circuit,

program 1

Taking the Mystery Out of Weaning the Pediatric Patient from the Ventilator

Peter Betit, BS, RRT, FAARC, and Richard D. Branson, BA, RRT, FAARC

Learn when to begin the process and how to recognize critical events in weaning a pediatric patient. Also teaches the physiological differences between the adult and pediatric patient and why weaning of the pediatric patient is different. The presentation confronts participants with options in providing assisted ventilation and the correct selection of options that expedite weaning

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program 2

Pulmonary Rehabilitation: Standard Care for Chronic Lung Disease Patients

Trina Limberg, BS, RRT, and Thomas J. Kallstrom, RRT, FAARC

Presentation details when to refer a patient for pulmonary rehabilitation and the four elements necessary for the successful operation of a rehabilitation service. Details how to prepare a treatment plan during assessment and how to modify it based on subsequent evaluations as well as how to incorporate rehabilitation techniques into routine bedside therapy sessions

Videotape available

program 3

Noninvasive Ventilation: The Latest Word

Dean R. Hess, PhD, RRT, FAARC, and Richard D. Branson, BA, RRT, FAARC

Learn how to avoid intubation in the acutely ill patient through identification of patients most likely to benefit from noninvasive ventilation. Learn selection and proper fit of full masks or nasal masks and how to select the proper ventilator based on the patient's condition and desired outcomes. Also learn when to make adjustments to achieve the goals of unloading respiratory muscles and achieving good patient/ventilator synchrony

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April 24, 11:30 a.m. - 1:00 p.m. Central Time
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May 29, 11:30 a.m. - 12:00 Noon Central Time

program 4

Education of the Patient with Asthma

Tracey Mitchell, RRT, RPFT, and Thomas J. Kallstrom, RRT, FAARC

This program teaches how to ensure that patients understand the disease process of asthma and their care plan for effective disease management. And, it details what patient education materials are available, their content, where to find them, and the best methods of presentation, including new terminologies, analogies, and techniques

Sponsored in part by an educational grant from Sepracor, Inc

Live Videoconference
May 22, 11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape
June 19, 11:30 a.m. - 12:00 Noon Central Time

program 5

ARDS: The Disease and Its Management

Leonard D. Hudson, MD, and David J. Pierson, MD, FAARC

Presents the four diagnostic criteria for ARDS and the six clinical risk factors that place patients at increased likelihood for developing ARDS. The program will teach viewers how to understand the implications of the lower and upper inflection points on the pressure-volume curve of the respiratory system in ARDS patients; and instruct them in the calculation of estimated required tidal volume

Live Videoconference -
June 26, 11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape -
July 17, 11:30 a.m. - 12:00 Noon Central Time

program 6

New Respiratory Drugs: What, When, and How?

Joseph L. Rau, PhD, RRT, FAARC, and Patrick J. Dunne, MEd, RRT, FAARC

Introduces participants to new formulations such as racemic drug mixtures and single isomers and their effective duration and how they lead to lower costs with improved patient responses. Viewers will learn the use of improved anticholinergics in the treatment of asthma patients and learn the uses and effects of inhaled anti-infective agents

Sponsored in part by an educational grant from Sepracor, Inc.

Live Videoconference
Aug.14, 11:30 a.m. - 1:00 p.m. Central Time
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Sept.11, 11:30 a.m. - 12:00 Noon Central Time

program 7

Invasive Ventilation: The Latest Word

Richard Kallet, MS, RRT and Richard D. Branson, BA, RRT FAARC

Learn how proper ventilator management can preclude inflicting harm on the patient and why it is essential for the clinician to understand the function and mechanics of newer mechanical ventilators. Also learn how reducing the patient's work of breathing is essential in reducing the additional load on ventilatory musculature, and why reinflating lungs and enhancing the functional area of the lung demands extraordinary means

Live Videoconference -
Sept. 25, 11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape -
Oct. 16, 11:30 a.m. - 12:00 Noon Central Time

program 8

Test Your Lungs, Know Your Numbers, Prevent Emphysema

Thomas L. Petty, MD, FAARC and David J. Pierson, MD, FAARC

Reviews the classic signs of COPD with an emphasis on emphysema and a discussion on the measures used to relieve symptoms and slow disease progression. Covers the importance of pulmonary function tests to determine VC, FFC, and FEV1; and why getting patients to know their numbers is the key to early diagnosis and successful treatment

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and the modes of ventilation were introduced in a randomized manner. MEASUREMENTS AND MAIN RESULTS: We measured visual analog scores for comfort for the three modes of ventilation and collected a ranking order and open-ended comments. We demonstrated that at the level of support we imposed, assisted spontaneous breathing was the most comfortable mode of ventilation and that synchronized intermittent mandatory ventilation was the most uncomfortable. These results were strongly supported by both the ranking scale and comments of the volunteers. CONCLUSIONS: Assisted spontaneous breathing was the most comfortable mode of ventilation because the pattern was primarily determined by the volunteer. Synchronized intermittent mandatory ventilation was the most uncomfortable because the ventilatory pattern was imposed on the volunteers, leading to ventilator-volunteer dyssynchrony. We also conclude there is wide individual variation in the subjective feeling of comfort. Whereas the mode of ventilation in ICUs is based primarily on the physiologic needs of the patient, the feeling of comfort may be considered when choosing an appropriate mode of ventilation during the weaning phase of critical illness.

Estimating Cardiac Filling Pressure in Mechanically Ventilated Patients with Hyperinflation —Teboul JL, Pinsky MR, Mercat A, Anguel N, Bernardin G, Achard JM, et al. Crit Care Med 2000 Nov;28(11):3631-3636.

OBJECTIVE: When positive end-expiratory pressure (PEEP) is applied, the intracavitary left ventricular end-diastolic pressure (LVEDP) exceeds the LV filling pressure because pericardial pressure exceeds 0 at end-expiration. Under those conditions, the LV filling pressure is itself better reflected by the transmural LVEDP (tLVEDP) (LVEDP minus pericardial pressure). By extension, end-expiratory pulmonary artery occlusion pressure (eePAOP), as an estimate of end-expiratory LVEDP, overestimates LV filling pressure when pericardial pressure is >0, because it occurs when PEEP is present. We hypothesized that LV filling pressure could be measured from eePAOP by also knowing the proportional transmission of alveolar pressure to pulmonary vessels calculated as index of transmission = (end-inspiratory PAOP–eePAOP)/(plateau pressure–total PEEP). We calculated transmural pulmonary artery occlusion pressure (tPAOP) with this equation: tPAOP = eePAOP–(index of transmission × total PEEP). We compared tPAOP with airway disconnection nadir PAOP measured during rapid airway disconnection in subjects undergoing PEEP with and without evidence of dynamic pulmonary hyperinflation. DESIGN: Prospective study. SETTING: Medical intensive care unit of a university hospital. PATIENTS: We studied 107 patients mechanically ventilated with PEEP for acute respiratory failure. Patients without dynamic pulmonary hyperinflation (group A, n = 58) were analyzed separately from patients with dynamic pulmonary hyperinflation (group B, n = 49). INTERVENTION: Transient airway disconnection. MEASUREMENTS AND MAIN RESULTS: In group A, tPAOP (8.5±6.0 mm Hg) and nadir PAOP (8.6±6.0 mm Hg) did not differ from each other but were lower than eePAOP (12.4±5.6 mm Hg, p < 0.05). The agreement between tPAOP and nadir PAOP was good (bias, 0.15 mm Hg; limits of agreement, -1.5-1.8 mm Hg). In group B, tPAOP (9.7±5.4 mm Hg) was lower than both nadir PAOP and eePAOP (12.1±5.4 and 13.9±5.2 mm Hg, respectively, p < 0.05 for both comparisons). The agreement between tPAOP and nadir PAOP was poor (bias, 2.3 mm Hg; limits of agreement, -0.2-4.8 mm Hg). CONCLUSIONS: Indexing the transmission of proportional alveolar pressure to PAOP in the estimation of LV filling pressure is equivalent to the nadir method in patients without dynamic pulmonary hyperinflation and may be more reliable than the nadir PAOP method in patients with dynamic pulmonary hyperinflation.

Intensive Care Unit Drug Use and Subsequent Quality of Life in Acute Lung Injury Patients —Nelson BJ, Weinert CR, Bury CL, Marinelli WA, Gross CR. Crit Care Med 2000 Nov;28(11):3626-3630.

OBJECTIVE: To examine the relationship between the use of sedative and neuromuscular blocking agents during a patient's intensive care unit (ICU) stay and subsequent measures of health-related quality of life. DESIGN: Cross-sectional mail survey and retrospective medical record abstraction of a prospectively identified cohort of lung injury patients. SETTING: ICUs in three teaching hospitals in a major metropolitan area. PATIENTS: Patients with acute lung injury (n = 24). INTERVENTIONS: None; observational study. MEASUREMENTS AND MAIN RESULTS: Patients' charts were reviewed for those patients returning postdischarge quality-of-life questionnaires. Duration, daily dose, and route of administration for sedatives and neuromuscular blocking agents were abstracted from ICU flow sheets. Relationships among ICU variables (days of sedation, days of neuromuscular blockade, and severity of illness as measured by Acute Physiology and Chronic Health Evaluation III score) and outcomes (symptoms of depression and symptoms of posttraumatic stress disorder) were assessed. Depressive symptoms at follow-up were correlated with days of sedation (p = 0.007), but not with days of neuromuscular blockade or initial severity of illness. The composite posttraumatic stress disorder symptom impact score was correlated with days of sedation (p = 0.006) and days of neuromuscular blockade (p = 0.035), but not with initial severity of illness. There were no significant differences between the frequency of patients reporting a specific posttraumatic stress disorder symptom in the high sedation group and the low sedation group, and there were no significant differences in specific posttraumatic stress disorder symptoms between the group that had received neuromuscular blockade and those who had not. CONCLUSIONS: The use of sedatives and neuromuscular blocking agents in the ICU is positively associated with subsequent measures of depression and posttraumatic stress disorder symptoms 6-41 months after ICU treatment for acute lung injury.

Longitudinal Study of Pediatric House Officers' Attitudes Toward Death and Dying —Vazirani RM, Slavin SJ, Feldman JD. Crit Care Med 2000 Nov;28(11):3740-3745.

OBJECTIVE: To investigate pediatric residents' attitudes toward end-of-life issues and their education in dealing with these issues. DESIGN: Exploratory survey. SETTING: Department of Pediatrics at the University of California, Los Angeles, Center for Health Sciences. SUBJECTS: Volunteer sample. A total of 182 of 203 pediatric residents at all levels of training completed anonymous questionnaires. INTERVENTIONS: None. MEASUREMENTS AND MAIN RESULTS: Data on residents' attitudes toward issues of death and dying and the efficacy of educational interventions were collected over a 4-yr period. When entering training, house officers are uncomfortable dealing with death and dying issues (mean, 3.3 of 5; 5 = not comfortable). By the end of their training, these house officers become comfortable dealing with these issues (mean, 2.2; p < 0.05). During their first 2 yrs of training, house officers report that their medical education is not helping them to deal with the issues of death and dying (mean, 3.3). At the end of their third year of training, residents report that their education is helping them to deal with these issues (mean, 2.5, p < 0.05). Strikingly, as house officers progress through their residency, they become less comfortable with the idea of administering pain medication to a dying patient, because the pain medication might hasten the patient's death (p < 0.05). CONCLUSIONS: Pediatric residents may benefit from more formal training in the practical aspects of death and dying issues. Residency education should do more to address these issues systematically for the benefit of both the residents and the patients and family members.

Role of Mouth-to-Mouth Rescue Breathing in Bystander Cardiopulmonary Resuscitation for Asphyxial Cardiac Arrest —Berg RA. Crit Care Med 2000 Nov;28(11 Suppl):N193-N195.

There is increasing evidence that mouth to mouth rescue breathing may not be necessary during brief periods of bystander cardiopulmonary resuscitation (CPR) for ventricular fibrillation. In contrast to ventricular fibrillation cardiac arrests, it has been assumed that rescue breathing is essential for treatment of asphyxial cardiac arrests because the cardiac arrests result from inadequate ventilation. This review explores the role of mouth-to-mouth rescue breathing during bystander CPR for asphyxial cardiac arrests. Clinical data suggest that survival from apparent asphyxial cardiac arrest can occur after CPR consisting of chest compressions alone, without rescue breathing. Two randomized, controlled swine investigations using models of bystander CPR for asphyxial cardiac arrest establish the following: a) that prompt initiation of bystander CPR is a crucially important intervention, and b) that chest compressions plus mouth-to-mouth rescue breathing is markedly superior to either technique alone. One of these studies further demonstrates that early in the asphyxial pulseless arrest process doing something (mouth-to-mouth rescue breathing or chest compressions) is better than doing nothing.

Cardiopulmonary Resuscitation without Ventilation—Kern KB. Crit Care Med 2000 Nov;28(11 Suppl):N186-N189

Current resuscitation methods, although occasionally effective, rarely perform as well as initially anticipated. Some of the disappointment can be attributed to the difficulty of the task for many, including both professional and lay first responders. Significant attention has been paid recently to the need to simplify both the technique and the teaching of resuscitation. In considering simplification of the current resuscitation scheme, a logical start is an honest reappraisal of the importance and priorities of each of the once sacrosanct ABCs, specifically, establishment of an Airway, artificial Breathing (mouth-to-mouth breathing), and chest compressions for temporary Circulation. Experimental data continue to accumulate indicating that most important within this triad is circulation. Adequate oxygen exists within the blood during at least the first 10 mins of cardiac arrest. If circulation is provided to distribute such oxygen, no survival disadvantage results with chest compression-only basic life support (BLS) efforts. Even a totally occluded airway during the first 6 mins of cardiac arrest does not compromise survival if reasonable circulation is provided with chest compressions. Clinical studies support the same conclusion that what most influences survival in any BLS effort is circulation, not ventilation. Belgium investigators have shown equal survival rates among those treated with chest compressions plus ventilation and those who received chest compressions alone. Telephone dispatcher-guided BLS cardiopulmonary resuscitation (CPR) has likewise shown no survival disadvantage to chest compression-only CPR when compared with telephone-guided standard BLS CPR. Based on this reasoning, a new simplified BLS method has been proposed. "Staged" CPR consists of a strategy to initially teach laypersons a simplified approach to BLS, which requires only chest compressions and not mouth-to-mouth breathing. "Bronze" CPR, in which chest compression-only BLS is taught, was compared with the standard European Resuscitation Council BLS course for laypersons. Manikin "exit testing" at course completion has revealed significant advantages of the simplified approach compared with standard CPR courses for the lay public.

Improving the Efficiency of Cardiopulmonary Resuscitation with an Inspiratory Impedance Threshold Valve—Lurie K, Zielinski T, McKnite S, Sukhum P. Crit Care Med 2000 Nov;28(11 Suppl):N207-N209

In an effort to improve the efficiency of cardiopulmonary resuscitation (CPR), a new inspiratory impedance threshold valve has been developed to enhance the return of blood to the thorax during the chest decompression phase. This new device enhances negative intrathoracic pressure during chest wall recoil or the decompression phase, leading to improved vital organ perfusion during both standard CPR and active compression decompression CPR. With active compression-decompression CPR, addition of the impedance threshold valve result... ...static ...ble pressures of ...55 mm Hg in patients in cardiac arrest. The ... shows promise for patients in asystole or shock-refractory ventricular fibrillation, when enhanced return of blood flow to the chest is needed to "prime the pump." The potential long-term benefits of this new valve remain under study.

Spontaneous Hemopneumothorax in Women—Kiser AC, Roberts CS. South Med J 2000 Dec;93(12):1209-1211

Spontaneous hemopneumothorax is uncommon, especially among women. We report a case of spontaneous hemopneumothorax in a 19-year-old woman and review seven other cases of spontaneous hemopneumothorax in women that have been reported in the English language.

Improvement of Sleep Apnea in Patients with Chronic Renal Failure Who Undergo Nocturnal Hemodialysis—Hanly PJ, Pierratos A. N Engl J Med 2001 Jan 11;344(2):102-107

BACKGROUND: Sleep apnea is common in patients with chronic renal failure and is not improved by either conventional hemodialysis or peritoneal dialysis. With nocturnal hemodialysis, patients undergo hemodialysis seven nights per week at home, while sleeping. We hypothesized that nocturnal hemodialysis would correct sleep apnea in patients with chronic renal failure because of its greater effectiveness. METHODS: Fourteen patients who were undergoing conventional hemodialysis for four hours on each of three days per week underwent overnight polysomnography. The patients were then switched to nocturnal hemodialysis for eight hours during each of six or seven nights a week. They underwent polysomnography again 6 to 15 months later on one night when they were undergoing nocturnal hemodialysis and on another night when they were not. RESULTS: The mean ($\pm$ SD) serum creatinine concentration was significantly lower during the period when the patients were undergoing nocturnal hemodialysis than during the period when they were undergoing conventional hemodialysis (3.9 ± 1.1 vs 12.8 ± 3.2 mg per deciliter [342 ± 101 vs 1131 ± 287 micromol per liter], p < 0.001). The conversion from conventional hemodialysis to nocturnal hemodialysis was associated with a reduction in the frequency of apnea and hypopnea from 25 ± 25 to 8 ± 8 episodes per hour of sleep (p < 0.03). This reduction occurred predominantly in seven patients with sleep apnea, in whom the frequency of episodes fell from 46 ± 19 to 9 ± 9 per hour (p < 0.006), accompanied by increases in the minimal oxygen saturation (from 89.2 ± 1.8 to 94.1 ± 1.6 percent, p < 0.005), transcutaneous partial pressure of carbon dioxide (from 38.5 ± 4.3 to 48.3 ± 4.9 mm Hg, p < 0.006), and serum bicarbonate concentration (from 23.2 ± 1.8 to 27.8 ± 0.8 mmol per liter, p < 0.001). During the period when these seven patients were undergoing nocturnal hemodialysis, the apnea-hypopnea index measured on nights when they were not undergoing nocturnal hemodialysis was greater than that on nights when they were undergoing nocturnal hemodialysis, but it still remained lower than it had been during the period when they were undergoing conventional hemodialysis (p < 0.05). CONCLUSIONS: Nocturnal hemodialysis corrects sleep apnea associated with chronic renal failure.

Coffee Consumption and the Risk of Coronary Heart Disease and Death—Kleemola P, Jousilahti P, Pietinen P, Vartiainen E, Tuomilehto J. Arch Intern Med 2000 Dec 11;160(22):3393-3400

OBJECTIVES: To study prospectively the relation of coffee drinking with fatal and nonfatal coronary heart disease (CHD) and all-cause mortality and to perform a cross-sectional analysis at baseline on the association between coffee drinking and CHD risk factors, diagnosed diseases, self-reported symptoms, and use of medicines. METHODS: The study cohort consisted of 20,179 randomly selected eastern Finnish men and women aged 30 to 59 years who participated in a cross-sectional risk factor survey in 1972, 1977, or 1982. Habitual coffee drinking, health

behavior, major known CHD risk factors, and medical history were assessed at the baseline examination. Each subject was followed up for 10 years after the survey using the national hospital discharge and death registers. Multivariate analyses were performed by using the Cox proportional hazards model. RESULTS: In men, the risk of nonfatal myocardial infarction was not associated with coffee drinking. The age-adjusted association of coffee drinking was J-shaped with CHD mortality and U-shaped with all-cause mortality. The highest CHD mortality was found among those who did not drink coffee at all (multivariate adjusted). Also, in women, all-cause mortality decreased by increasing coffee drinking. The prevalence of smoking and the mean level of serum cholesterol increased with increasing coffee drinking. Non-coffee drinkers more often reported a history of various diseases and symptoms, and they also more frequently used several drugs compared with coffee drinkers. CONCLUSIONS: Coffee drinking does not increase the risk of CHD or death. In men, slightly increased mortality from CHD and all causes in heavy coffee drinkers is largely explained by the effects of smoking and a high serum cholesterol level.

Temporal Trends in Outcomes of Older Patients with Pneumonia —
Metersky ML, Tate JP, Fine MJ, Petrillo MK, Meehan TP. Arch Intern Med 2000 Dec 11;160(22):3385-3391.

BACKGROUND: It is unclear how outcomes of care for patients hospitalized for pneumonia have changed as patterns of health care delivery have changed during the 1990s. This study was performed to determine trends in outcomes of care for older patients hospitalized for pneumonia. METHODS: This retrospective analysis was based on Medicare claims and included most patients with pneumonia who were older than 65 years and admitted to acute care hospitals in Connecticut between October 1, 1991, and September 30, 1997 (fiscal years 1992-1997). We assessed the trends in hospital costs, discharge destination, hospital mortality rates, mortality rates within 30 days of discharge, and 30-day readmission rates for pneumonia. Multivariate logistic regression analyses were used to adjust for differences in patient characteristics. RESULTS: The mean ($\pm$ SD) length of stay declined from 11.9 $\pm$ 11.4 days to 7.7 $\pm$ 7.2 days between 1992 and 1997. During this period, adjusted in-hospital mortality rates declined (p =0.02), while the adjusted risk of discharge to a nursing facility increased (p= 0.001) and the adjusted risk of hospital readmission for pneumonia within 30 days of discharge increased (p =0.05). The adjusted risk of death 30 days after discharge increased, although the difference was not statistically significant (p =0.09). CONCLUSIONS: Between 1992 and 1997, the adjusted risks of mortality after discharge, placement in a nursing facility, and hospital readmission for pneumonia increased among older patients hospitalized for pneumonia, in association with a decline in mean hospital length of stay. These findings raise the question of whether the declining hospital length of stay has negatively affected patient outcomes.

Out-of-Hospital Cardiac Arrest in Octogenarians and Nonagenarians — Kim C, Becker L, Eisenberg MS. Arch Intern Med 2000 Dec 11;160(22):3439-3443.

BACKGROUND: Studies of elderly patients who have out-of-hospital cardiac arrest have contradictory results. The studies usually define elderly patients as those older than 70 years, and include relatively few octogenarians and nonagenarians. OBJECTIVES: To compare the survival after out-of-hospital cardiac arrest of octogenarians, nonagenarians, and younger patients and to determine the influence of age on survival after adjusting for factors known to influence out-of-hospital cardiac arrest outcome. METHODS: We conducted a retrospective cohort study in suburban King County, Washington, on 5882 patients who had out-of-hospital cardiac arrest from presumed cardiovascular disease between January 1, 1987, and December 31, 1998, and who received cardiopulmonary resuscitation from bystanders, emergency medical technicians, or

both. The main outcome measure was survival to hospital discharge. RESULTS: In patients who had out-of-hospital cardiac arrest due to a cardiac cause, younger patients had higher hospital discharge rates than octogenarians, who in turn had higher hospital discharge rates than nonagenarians (19.4% vs 9.4% vs 4.4%; p<0.001). However, survival to hospital discharge improved significantly for younger patients, octogenarians, and nonagenarians who had ventricular fibrillation or pulseless ventricular tachycardia (36% vs 24% vs 17%; p<0.001). After multiple logistic regression analysis controlling for other factors, increased age was weakly associated with decreased survival to hospital discharge (odds ratio, 0.92; 95% confidence interval, 0.85-0.99). CONCLUSIONS: Octogenarians and nonagenarians have lower survival to hospital discharge than younger patients, but age is a much weaker predictor of survival than other factors such as initial cardiac rhythm. Decisions regarding resuscitation should not be based on age alone.

The Pathogenesis of Acute Pulmonary Edema Associated with Hypertension—Gandhi SK, Powers JC, Nomeir AM, Fowle K, Kitzman DW, Rankin KM, Little WC. N Engl J Med 2001 Jan 4;344(1):17-22.

BACKGROUND: Patients with acute pulmonary edema often have marked hypertension but, after reduction of the blood pressure, have a normal left ventricular ejection fraction ($\geq$ 0.50). However, the pulmonary edema may not have resulted from isolated diastolic dysfunction but, instead, may be due to transient systolic dysfunction, acute mitral regurgitation, or both. METHODS: We studied 38 patients (14 men and 24 women; mean [$\pm$SD] age, 67 $\pm$ 13 years) with acute pulmonary edema and systolic blood pressure > 160 mm Hg. We evaluated the ejection fraction and regional function by two-dimensional Doppler echocardiography, both during the acute episode and one to three days after treatment. RESULTS: The mean systolic blood pressure was 200 $\pm$ 26 mm Hg during the initial echocardiographic examination and was reduced to 139 $\pm$ 17 mm Hg (p<0.05) at the time of the follow-up examination. Despite the marked difference in blood pressure, the ejection fraction was similar during the acute episode (0.50 $\pm$ 0.15) and after treatment (0.50 $\pm$ 0.13). The left ventricular regional wall-motion index (the mean value for 16 segments) was also the same during the acute episode (1.6 $\pm$ 0.6) and after treatment (1.6 $\pm$ 0.6). No patient had severe mitral regurgitation during the acute episode. Eighteen patients had a normal ejection fraction (at least 0.50) after treatment. In 16 of these 18 patients, the ejection fraction was at least 0.50 during the acute episode. CONCLUSIONS: In patients with hypertensive pulmonary edema, a normal ejection fraction after treatment suggests that the edema was due to the exacerbation of diastolic dysfunction by hypertension – not to transient systolic dysfunction or mitral regurgitation.

Prehospital Intubation in Patients with Severe Head Injury —Murray JA, Demetriades D, Berne TV, Stratton SJ, Cryer HG, Bongard F, et al. J Trauma 2000 Dec;49(6):1065-1070.

BACKGROUND: Prehospital intubation and airway control is routinely performed by paramedics in critically injured patients. Despite the advantages provided by this procedure, numerous potential risks exist when this is performed in the field. We reviewed the outcome of patients with severe head injury, to determine whether prehospital intubation is associated with an improved outcome. METHODS: A retrospective review of registry data of patients admitted to an urban trauma center with severe head injury (field Glasgow Coma Scale score of < 8 and head Abbreviated Injury Scale score of >3) was performed. Patients were stratified by methods of airway control performed by prehospital personnel: not intubated, intubated, or unsuccessful intubation. Mortality was determined for each group. To control for significant variables between these populations, matching and multivariate analysis were performed. RESULTS: Patients requiring prehospital intubation or in whom intubation was attempted had an increased mortality (81% and 77%, respectively) when

compared with nonintubated patients (48%). The mortality for patients who had prehospital intubation performed did not demonstrate an improved survival using matching. In fact, intubated patients had a significantly higher relative risk (RR) of mortality when compared with nonintubation (RR = 1.74, p = 0.001) and unsuccessful intubation patients (RR = 1.53, p = 0.008). CONCLUSION For patients with severe head injury, prehospital intubation did not demonstrate an improvement in survival. Further prospective randomized trials are necessary to confirm these results.

An Evidence-Based Cost-Effectiveness Model on Methods of Prevention of Posttraumatic Venous Thromboembolism—Velmahos GC, Oh Y, McCombs J, Oder D—J Trauma 2000 Dec;49(6):1059-1064

BACKGROUND Venous thromboembolism (VT) after injury is a major health problem. Literature data on methods of VT prophylaxis are not consistent with regard to safety and efficacy, and a recent evidence-based report could not conclude that any method was superior to any other or to no prophylaxis. Because no study exists on the cost-effectiveness (C-E) of the different methods of prophylaxis, data from the evidence-based report were used to design a C-E analysis. This analysis will assist in the design of future randomized trials with adequate power to show significant outcome differences. METHODS A decision-tree model was designed on the basis of outcomes from the evidence-based report or relevant literature. We then calculated the cost of prevention of VT by one of the most commonly used methods: low-dose heparin (LDH), low-molecular-weight heparin (LMWH), or sequential compression devices (SCDs) using different probabilities of incidence of VT. Finally, we adjusted the cost for expected years of life after the episode of VT to calculate the cost per life-year saved by preventing VT. RESULTS We produced two tables that can be used to calculate the cost per life-year saved for any patient according to his or her age and the method of prophylaxis used. VT prophylaxis becomes less cost-effective as age progresses, because of decreased life-expectancy. With a widely accepted cost limit of $50,000 per life-year saved to indicate cost-effective treatment, LDH is more cost-effective than LMWH or SCDs. CONCLUSION Our C-E model can help future investigators plan VT-related research with appropriate sample sizes to evaluate cost-effective methods of prophylaxis. LMWH and SCDs must demonstrate substantial improvements in measured outcomes to be more cost-effective than LDH. C-E must be incorporated as a primary outcome in future studies comparing different methods of VT prophylaxis.

A Survey of Physician Attitudes and Practices Concerning Cost-Effectiveness in Patient Care—Ginsburg ME, Kravitz RL, Sandberg WA—West J Med 2000 Dec;173(6):390-394

OBJECTIVE To identify physicians' views regarding cost-containment and cost-effectiveness and their attitudes and experience using cost-effectiveness in clinical decision making. DESIGN A close-ended 30-item written survey. SUBJECTS 1,000 randomly selected physicians whose practices currently encompass direct patient care and who work in the California counties of Sacramento, Yolo, Placer, Nevada, and El Dorado. Outcome measures Physician attitudes about the role of cost and cost-effectiveness in treatment decisions, perceived barriers to cost-effective medical practice, and response of physicians and patients if there are conflicts about treatment that physicians consider either not indicated or not cost-effective. RESULTS Most physicians regard cost-effectiveness as an appropriate component of clinical decisions and think that only the treating physician and patient should decide what is cost-worthy. However, physicians are divided on whether they have a duty to offer medical interventions with remote chances of benefit regardless of cost, and they vary considerably in their interactions with patients when cost-effectiveness is an issue. CONCLUSION Although physicians in the Sacramento region accept cost-effectiveness as important and appropriate in clinical

practice there is little uniformity in how cost-effectiveness is being implemented.

Increasing Prevalence of Multidrug-Resistant *Streptococcus pneumoniae* in the United States—Whitney CG, Farley MM, Hadler J, Harrison LH, Lexau C, Reingold A, et al—N Engl J Med 2000 Dec 28;343(26):1917-1924

BACKGROUND The emergence of drug-resistant strains of bacteria has complicated treatment decisions and may lead to treatment failures. METHODS We examined data on invasive pneumococcal disease in patients identified from 1995 to 1998 in the Active Bacterial Core Surveillance program of the Centers for Disease Control and Prevention. Pneumococci that had a high level of resistance or had intermediate resistance according to the definitions of the National Committee for Clinical Laboratory Standards were defined as "resistant" for this analysis. RESULTS During 1998, 4013 cases of invasive *Streptococcus pneumoniae* disease were reported (23 cases per 100,000 population); isolates were available for 3475 (87 percent). Overall, 24 percent of isolates from 1998 were resistant to penicillin. The proportion of isolates that were resistant to penicillin was highest in Georgia (33 percent) and Tennessee (35 percent), in children under five years of age (32 percent vs 21 percent for persons five or more years of age), and in whites (26 percent vs 22 percent for blacks). Penicillin-resistant isolates were more likely than susceptible isolates to have a high level of resistance to other antimicrobial agents. Serotypes included in the 7-valent conjugate and 23-valent pneumococcal polysaccharide vaccines accounted for 78 percent and 88 percent of penicillin-resistant strains, respectively. Between 1995 and 1998 (during which period 12,045 isolates were collected), the proportion of isolates that were resistant to three or more classes of drugs increased from 9 percent to 14 percent; there also were increases in the proportions of isolates that were resistant to penicillin (from 21 percent to 25 percent), cefotaxime (from 10 percent to 14 percent), meropenem (from 10 percent to 16 percent), erythromycin (from 11 percent to 15 percent), and trimethoprim-sulfamethoxazole (from 25 percent to 29 percent). The increases in the frequency of resistance to other antimicrobial agents occurred exclusively among penicillin-resistant isolates. CONCLUSIONS Multidrug-resistant pneumococci are common and are increasing. Because a limited number of serotypes account for most infections with drug-resistant strains, the new conjugate vaccines offer protection against most drug-resistant strains of *S pneumoniae*.

Pulmonary Hypertension—Gaine S—JAMA 2000 Dec 27;284(24):3160-3168

A clinically useful, treatment-based classification of pulmonary hypertension divides the disease into 5 distinct categories: (1) pulmonary hypertension associated with disorders of the respiratory system and/or hypoxemia, (2) pulmonary venous hypertension, (3) chronic thromboembolic disease, (4) pulmonary arterial hypertension, and (5) pulmonary hypertension due to disorders directly affecting the pulmonary vasculature. Pulmonary arterial hypertension includes individuals with primary pulmonary hypertension, congenital heart disease, connective tissue disease, and liver disease. These heterogeneous diseases have similar characteristic pathological changes, including in situ thrombosis, smooth muscle hypertrophy, and intimal proliferation. Right heart catheterization is essential to confirm diagnosis, determine prognosis, and assign therapy. A minority of patients have a favorable response to an acute vasodilator trial and long-term benefit with calcium channel blocker therapy. Continuous intravenous epoprostenol improves symptoms and survival in patients with advanced primary pulmonary hypertension and has potential benefit in other forms of pulmonary arterial hypertension. Lung transplantation remains an important option for individuals in whom maximal medical therapy fails. The recent discovery of the gene for familial primary pulmonary hypertension and the increase in new drugs undergoing clinical trials are encouraging developments.

Original Contributions

Effect of Inner Cannula Removal on the Work of Breathing Imposed by Tracheostomy Tubes: A Bench Study

Tony Cowan RRT CPFT, Timothy B Op't Holt EdD RRT, Cyndi Gegenheimer RRT, Seth Izenberg MD, and Pandurang Kulkarni PhD

BACKGROUND: Tracheotomy has been used to assist in weaning patients from mechanical ventilation. Some patients fail to be weaned from the ventilator despite tracheostomy. We hypothesized that removing the inner cannula from the tracheostomy tube would decrease the tube's imposed work of breathing (WOB_{IMP}). METHODS: The hypothesis was tested using a lung model, by measuring the change in WOB_{IMP} when the inner cannula was removed. A mechanical lung model was developed using a test lung to simulate a spontaneously breathing patient. WOB_{IMP} was measured with a commercially available lung mechanics monitor. Shiley size 6, 8, and 10 nonfenestrated tracheostomy tubes were tested with the inner cannula in and out. Breathing conditions were simulated using tidal volumes (V_T) of 300 and 500 mL matched with breathing frequencies of 12, 24, and 32 breaths per minute, by using a ventilator to simulate spontaneous breathing through one side of the test lung. RESULTS: Under all the tested breathing conditions, WOB_{IMP} for each of the 3 tracheostomy tubes was significantly reduced ($p < 0.05$) when the inner cannula was removed. Also, as simulated spontaneous inspiratory flow demand increased (ie, as V_T and/or frequency were increased), WOB_{IMP} also increased, and vice versa. With the cannula removed, WOB_{IMP} was not significantly different between the size 6 and 8 tubes nor between the size 8 and 10 tubes when V_T was 300 mL and frequency was 12 breaths per minute. CONCLUSIONS: There was a significant decrease in WOB_{IMP} with each tube when the inner cannula was removed. WOB_{IMP} increased with an increase in inspiratory flow demand (ie, increase in V_T and/or frequency), as well as when tube size decreased. In weaning a tracheostomized patient from mechanical ventilation, increasing the internal diameter of the tube by removing the inner cannula may be beneficial. Further study is needed to determine if these findings are clinically important. *Key words: airway resistance, BiCore pulmonary monitor, pulmonary monitoring, tracheostomy, ventilator weaning, weaning, work of breathing, pulmonary mechanics.* [Respir Care 2001;46(5):460–465]

Background

Tracheotomy is a common surgical procedure for intensive care patients.[1] The goals of tracheotomy are to bypass the upper airway, facilitate removal of tracheobronchial secretions, prevent aspiration of gastric contents, and to control the airway for prolonged mechanical ventilation.[1-4] Despite known disadvantages of tracheotomy, which include tracheal stenosis at the stoma site, increased bacterial colonization of the airway, and prolonged tracheal cannulation, tracheostomy tubes provide a number of advantages over endotracheal tubes.[5,6] In the event of prolonged ventilation, tracheostomy tubes provide improved patient mobility and comfort, improved secretion clearance, increased airway security, relief from worsening glot-

Fig. 1. Lung model used to measure imposed resistive work of breathing in tracheostomy tubes with the cannula in and out. TTL = training/test lung.

tic and subglottic stenosis, relief from worsening oropharyngeal and laryngeal damage, and perhaps fewer days of mechanical ventilation.[1,5-8] Ventilator-dependent patients better tolerate weaning with tracheostomy tubes than endotracheal tubes, because tracheostomy offers lower airway resistance and up to 50% less dead space, making spontaneous breathing considerably easier.[4,6,8]

We have observed failure to wean from ventilation in some patients who have tracheostomies. If a method can be introduced to promote successful weaning, more patients may be liberated from mechanical ventilation faster and with less stress.

A comparison of the imposed resistive work of breathing (WOB) of a tracheostomy tube with the inner cannula in place and with the inner cannula removed has not previously been reported. The difference in the imposed WOB (WOB_{IMP}) between these two conditions needed to be measured to determine if removing the inner cannula would lower WOB_{IMP}. The purpose of this study was to determine if removal of the inner cannula of a tracheostomy tube in a lung model causes a significant decrease in WOB_{IMP}.

Materials and Methods

A mechanical model of the lung and airway (Fig. 1) was assembled using a commercially available training/test lung (2600i PneuView Dual Adult Testing and Training System, Michigan Instruments, Grand Rapids, Michigan). Shiley adult tracheostomy tubes (Mallinckrodt, Irvine, California) were connected to the right side of the 2-chamber test lung by inserting the distal end of the tracheostomy tube into an adapter that connected to the test lung's 15 mm connector. The tracheostomy tube cuff was inflated as needed to create an air-tight seal. A lift bar was attached to the left test lung chamber, which was ventilated by a time-cycled, volume-limited ventilator (Emerson 3MV-PED ventilator, JH Emerson, Cambridge, Massachusetts). The ventilator delivered tidal volumes (V_T) of 500 and 300 mL.

at frequencies of 12, 24, and 32 breaths per minute, with a sinusoidal inspiratory flow waveform. The inhalation-to-exhalation time ratio was maintained at 1:2 by setting the inspiratory time to 1.67 seconds, 0.84 second, and 0.63 second, respectively. Inspiratory flow varied with frequency, V_T, and inspiratory time. Each frequency was matched with the specified V_T to simulate 6 conditions of quiet, moderate, and labored breathing: V_T of 500 and 300 mL were paired with a frequency of 12 breaths per minute to simulate quiet breathing conditions; V_T of 500 and 300 mL were paired with a frequency of 24 breaths per minute to simulate moderate breathing conditions; and V_T of 500 and 300 mL were paired with a frequency of 32 breaths per minute to simulate labored breathing conditions. Each V_T and frequency was verified using a calibrated pulmonary mechanics monitor (BiCore CP-100, Allied Healthcare Products, St Louis, Missouri).

The pulmonary mechanics monitor was used to measure the airway pressure drop at the carinal (distal) end of the tracheostomy tube and V_T for calculation of WOB_{IMP}. This was done by attaching a 1 mm internal-diameter, air-filled silastic catheter, attached to the pulmonary mechanics monitor, to the distal end of the tracheostomy tube to measure the pressure drop (below baseline). Also, a flow transducer was attached to the proximal end of the tracheostomy tube to measure flow. The pulmonary mechanics monitor integrates flow, resulting in a V_T measurement.

The pulmonary mechanics monitor has a built-in balloon integrity test that is conducted before the monitor will allow any WOB measurements to be made. When using an esophageal balloon catheter in a patient, checking the integrity of the balloon is useful. If the balloon's integrity is compromised while in the esophagus, the accuracy of the pressure readings may be affected by partially or totally blocked pressure ports. However, in this model, the balloon would have hindered the nonesophageal pressure readings and was therefore removed. In order to bypass the balloon integrity test, we placed a 3-way stopcock in the pressure measurement line. The extension tubing from the monitor was cut and the severed end was connected to the proximal port of the stopcock. The connector of an esophageal balloon catheter was cut off, and the severed end of the balloon catheter was connected to the middle port of the stopcock. One end of the air-filled, silastic catheter was connected to the distal port of the stopcock, and the other end was put through an opening in the adapter near the distal end of the tracheostomy tube (see Fig. 1). The open area around the catheter in the adapter was sealed with silicone gel. This was similar to the method described by Banner et al,[9] to switch between esophageal pressure and carinal pressure when alternating between measurements of patient WOB and WOB_{IMP}, respectively. Instead of switching between 2 sites for pressure measurements, the stopcock was used to switch between the balloon catheter and
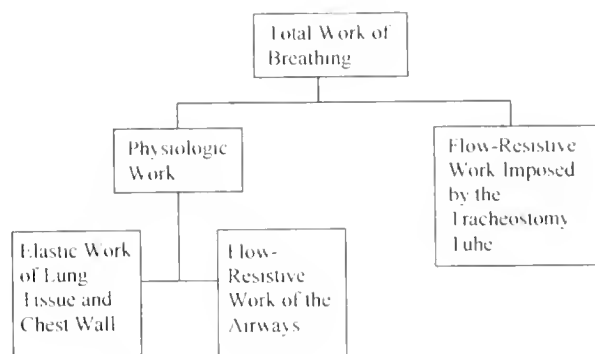
Fig. 2. In a spontaneously breathing patient with a tracheostomy tube, the total work of breathing is composed of physiologic work (including the elastic work of the lung and chest wall) and flow-resistive work imposed by the tracheostomy tube.

the silastic catheter used to measure the pressure drop at the distal end of the tracheostomy tube. The esophageal balloon catheter port was turned on when the monitor conducted the balloon integrity test. After the test was finished, the port to the silastic catheter was opened, allowing the monitor to receive pressure readings from the distal end of the tracheostomy tube.

Total WOB (WOB_{TOT}) is composed of physiologic WOB (WOB_{PHYS}), which includes elastic work of the lung tissue and chest wall and flow-resistive work of the airways, and WOB_{IMP}, which is the flow-resistive work of the breathing device (Fig. 2).[10] The WOB_{IMP} of the tracheostomy tube was calculated by using the following adapted equation:

$$WOB_{IMP} = \int P_{TT} \cdot dV$$

wherein P_{TT} is the pressure drop at the distal end of the tracheostomy tube and dV is the change in volume.[11] Shiley tracheostomy tube sizes 6, 8, and 10 were used in this study. During the portion of the study where the inner cannula was removed, the tracheostomy tube was attached to the flow transducer after removing the inner cannula from the 15 mm connector and reattaching the connector to the tracheostomy tube. Silicone gel was applied to the outside of the connector once it was reattached to the tracheostomy tube, to establish an airtight seal. Applying silicone-gel to the tracheostomy tube of a patient is not practical and could even prove hazardous. Although finding a safe method of attaching a flow transducer to a noncannulated tracheostomy tube proves complicated, we believe this obstacle can be overcome.

All measurements were made at room temperature. Fluctuations in barometric pressure and humidity were not taken into consideration. A constant test lung compliance of 0.04 L/cm H_2O was maintained throughout the study.
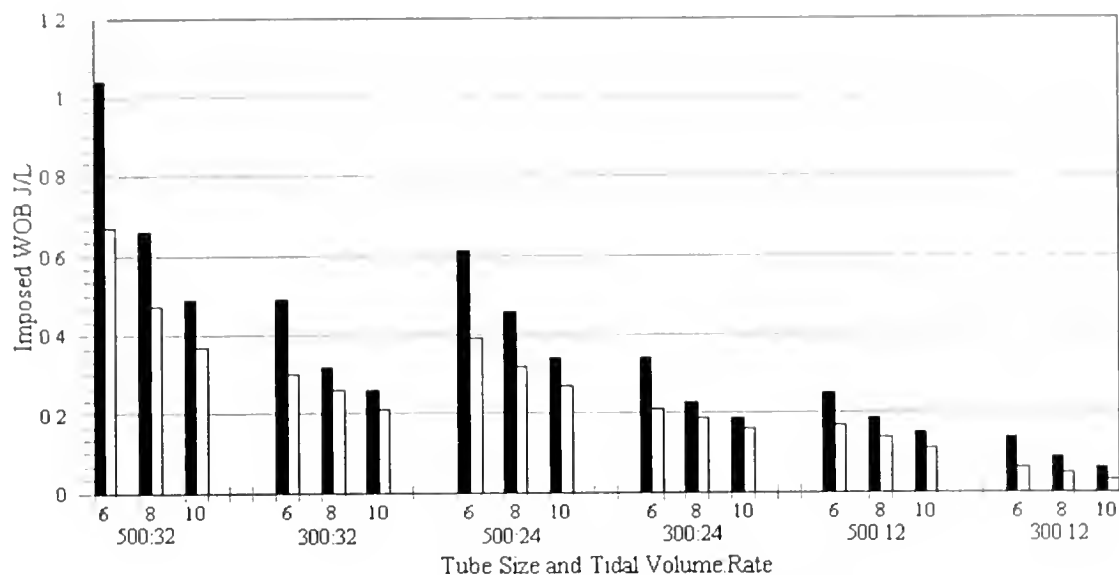
Fig. 3. Imposed work of breathing (WOB) for Shiley size 6, 8, and 10 tracheostomy tubes, with tidal volumes of 500 and 300 mL and respiratory rates of 12, 24, and 32 breaths per minute. Black bars denote WOB with the cannula in place. Clear bars denote WOB with the cannula removed

The WOB_{IMP} was measured for each tube, under each simulated breathing condition with the cannula in and out. This was achieved by using the pulmonary monitor's numeric data mode, which provided a breath-by-breath analysis of WOB. Unfortunately, similar to Blanch and Banner,[12] the pulmonary mechanics monitor we used did not directly measure WOB_{IMP}. Instead, patient WOB or WOB_{TOT} was measured and displayed on the monitor. Correction factors of 0.06 J/L per 500 mL of V_T (elastic work required to inflate the respiratory system) and 0.02 J/L per 300 mL of V_T were subtracted from each measured total WOB value to mathematically derive WOB_{IMP}. As reported by Blanch and Banner,[12] for the BiCore CP-100 respiratory monitor, a correction factor is needed to avoid overestimating the reported WOB_{IMP} for a model in which the elastic work of the chest wall is not a factor

In patients, the BiCore monitor uses the Campbell diagram to calculate WOB_{TOT}.[13] The BiCore reports WOB_{TOT} by integrating pressure drop and V_T, creating a pressure-volume loop. The area under the pressure-volume loop is the resistive and elastic work needed to inflate the lungs; however, it does not account for all of the elastic work needed to expand the entire respiratory system. To include the missing elastic work, the monitor uses a programmed chest wall compliance (200 mL/cm H_2O) and measured V_T to calculate the approximate missing elastic work, then adds that value to the work measured in the area of the pressure-volume loop. Since chest wall compliance was not a factor in our model, this added approximation for elastic WOB of the chest wall was subtracted. Otherwise, falsely high WOB_{IMP} values would have been reported.

The average WOB_{IMP} (corrected) from 10 consecutive breaths was used in calculating the results. The final mean WOB_{IMP} values within each test were compared statistically with 4-way analysis of variance. Since interactions were significant, multiple comparisons were analyzed using Tukey's Honest Significant Difference test, with an overall significance level of alpha ≤ 0.05. Using this method, any 2 means are declared significant if the absolute difference in the means is greater than 0.025 J/L.

Results

Imposed resistive WOB decreased significantly when the inner cannula was removed from Shiley size 6, 8, and 10 tracheostomy tubes under simulated quiet, moderate, and labored breathing conditions. Also, as inspiratory flow demand increased, imposed resistive WOB increased significantly with each tube (Fig. 3 and Table 1). During the lowest level breathing condition (300:12), there was an insignificant difference in WOB_{IMP} between the size 6 and 8 tubes with no cannula. This was also true for the size 8 and 10 tubes under the same conditions.

Discussion

Weaning patients from mechanical ventilation can be a challenging and sometimes cumbersome task. Patients with compromised pulmonary function may take weeks to liberate from mechanical ventilation. Fortunately for the patient, tracheostomy may facilitate weaning, decreasing the number of ventilator days.[1,4,6,8] Tracheostomy tubes offer

Table 1. Imposed Work of Breathing for Shiley Size 6, 8, and 10 Tracheostomy Tubes Under 3 Breathing Patterns

V_T:f		WOB$_{IMP}$ (J/L)		
		Shiley size 6	Shiley size 8	Shiley size 10
500:32	C	1.04 ± 0.007	0.66 ± 0.013	0.49 ± 0.005
	NC	0.67 ± 0.014	0.47 ± 0.007	0.37 ± 0.008
300:32	C	0.49 ± 0.005	0.32 ± 0.005	0.26 ± 0.003
	NC	0.30 ± 0.009	0.26 ± 0.007	0.21 ± 0.007
500:24	C	0.61 ± 0.003	0.46 ± 0.005	0.34 ± 0.008
	NC	0.39 ± 0.008	0.32 ± 0.000	0.27 ± 0.003
300:24	C	0.34 ± 0.005	0.23 ± 0.004	0.19 ± 0.003
	NC	0.21 ± 0.005	0.19 ± 0.005	0.16 ± 0.005
500:12	C	0.25 ± 0.006	0.19 ± 0.005	0.15 ± 0.003
	NC	0.17 ± 0.009	0.14 ± 0.007	0.11 ± 0.020
300:12	C	0.14 ± 0.007	0.09 ± 0.007	0.06 ± 0.006
	NC	0.06 ± 0.003	0.05 ± 0.009	0.03 ± 0.011

WOB$_{IMP}$ = imposed work of breathing
V_T = tidal volume
f = respiratory frequency
C = cannula
NC = no cannula
*WOB$_{IMP}$ for Shiley size 6, 8, and 10 tracheostomy tubes under 3 breathing patterns that simulate labored, moderate, and quiet breathing conditions. Means and standard deviations were calculated from 10 breaths obtained by the Bicore CP-100 monitor. Removal of the inner cannula resulted in a statistically significant ($p < 0.05$) decrease for each tube under each V_T:f combination. Any 2 means are declared significant if the absolute difference in the means is greater than 0.025 J/L, using Tukey's Honest Significant Difference test.

less dead space and airway resistance than endotracheal tubes, thereby lowering WOB and making spontaneous breathing easier.[4,8,14] We hypothesized that removal of the inner cannula would decrease WOB$_{IMP}$. We believe this would facilitate weaning in the clinical setting.

Although there is no literature addressing WOB$_{IMP}$ of tracheostomy tubes with the inner cannula removed, there have been studies conducted on WOB$_{IMP}$ of tracheostomy tubes and artificial airways in general. In one study comparing the imposed WOB of endotracheal and tracheostomy tubes in a lung model, the WOB$_{IMP}$ in a tracheostomy tube was found to be lower than in an endotracheal tube with the same internal diameter.[14] This was more pronounced with increasing flow rates. Increased resistance caused by increasing turbulent flow and longer tube length were suggested reasons for these findings. These observations were consistent with Poiseuille's law, which indicates that under certain conditions, changes in pressure vary directly with tube length and flow.

Bolder et al[15] measured WOB$_{IMP}$ with endotracheal tubes in a lung model and found a 34–154% increase in WOB with only a 1.0 mm decrease in internal diameter. Resistance may be even higher in patients with these tubes because of the addition of secretions and the thermolability of the plastic.[14]

Similarly, Mullins et al[16] measured the resistance and WOB in tracheostomy tubes and found that WOB decreased with increasing internal diameter of the tube. It was noted that increases in respiratory rate and V_T are essentially different methods of increasing flow and are directly associated with increased WOB. It was also suggested that, to facilitate weaning a tracheostomized patient from mechanical ventilation, selecting a tube that optimally lowers imposed WOB is critical for success.

In all the conditions considered, our results indicate that statistically significant reductions in imposed resistive WOB can be achieved by removal of the inner cannula. Even during the lowest simulated breathing condition, when the imposed WOB was minimal to start with, the reduction achieved by removing the inner cannula was significant. The reductions generally became more prominent as V_T and frequency increased. It was also clear that as the inspiratory flow demand decreased, a statistically significant decrease was observed in WOB$_{IMP}$. Furthermore, under each condition considered in our study, WOB$_{IMP}$ decreased significantly as the tube size was increased, except when V_T was 300 mL and frequency was 12 breaths per minute, which yielded an insignificant difference in WOB$_{IMP}$ between the size 6 and 8 tubes and the size 8 and 10 tubes, with the cannula removed.

The normal range of physiologic WOB in the adult patient is 0.3–0.6 J/L.[17] In patients on partial ventilatory support with WOB$_{PHYS}$ values greater than 0.75–0.80 J/L, weaning is unlikely to be successful.[17,18] Kirton et al[18] demonstrated that intubated patients with an unacceptably high WOB$_{TOT}$ (ie, 1.6 ± 0.83 J/L) and a WOB$_{PHYS}$ less than 0.80 J/L could be successfully extubated. WOB$_{IMP}$ was often twice that of WOB$_{PHYS}$, meaning that WOB$_{IMP}$ can masquerade as ventilator weaning intolerance. Unfortunately for the tracheostomized patient weaning from mechanical ventilation, complete decannulation is not an option to alleviate the hindering effects of WOB$_{IMP}$ of the tracheostomy tube. Therefore, it is our opinion that WOB$_{TOT}$ must be less than the acceptable WOB$_{PHYS}$ (ie, less than 0.75–0.80 J/L) to promote successful weaning in the tracheostomized patient. If resistive WOB imposed by the inner cannula can be eliminated during weaning to achieve WOB$_{TOT}$ values less than 0.75–0.80 J/L, weaning may be facilitated.

WOB$_{IMP}$ decreased as tube size increased and inspiratory flow demand (V_T or frequency) decreased, with and without the cannula. The hard-to-wean patient may initially exhibit a labored breathing pattern resulting in a greater than normal WOB$_{PHYS}$. In addition, if the patient has a small tracheostomy tube, high levels of WOB$_{IMP}$ would make weaning even more difficult and prevent success in weaning.

If a patient has a near but higher than normal WOB$_{PHYS}$ during weaning trials, the imposed WOB by the inner

cannula could cause weaning failure by increasing WOB$_{TOT}$ to greater than 0.75–0.80 J/L. This would especially hold true for the size 6 and 8 tubes under certain conditions. When V_T was 500 mL and frequency was 32 breaths per minute, removing the inner cannula significantly reduced the WOB$_{IMP}$ by 36% (0.67 J/L vs 1.04 J/L) with the size 6 tube; by 28% (0.47 J/L vs 0.66 J/L) with the size 8 tube; and by 24% (0.37 J/L vs 0.49 J/L) with the size 10 tube. Although the WOB$_{IMP}$ remained high (0.67 J/L) with the size 6 tube after cannula removal, a patient exhibiting a low WOB$_{PHYS}$ but a high WOB$_{TOT}$ due to a high WOB$_{IMP}$ may achieve an acceptable WOB$_{TOT}$ for weaning after cannula removal. Similarly, when V_T was 500 mL and frequency was 24 breaths per minute, the WOB$_{IMP}$ decreased significantly after inner cannula removal: by 36% (0.39 J/L vs 0.61 J/L) with the size 6 tube; by 30% (0.32 J/L vs 0.46 J/L) with the size 8 tube; and by 21% (0.27 J/L vs 0.34 J/L) with the size 10 tube. The WOB$_{IMP}$ was practically the same for the size 8 and 10 tubes with the inner cannula in place when V_T was 300 mL and frequency was 12 breaths per minute. This indicates the potential that removing the inner cannula can decrease WOB$_{TOT}$ to an acceptable range for promoting weaning, by decreasing WOB$_{IMP}$.

The tracheostomy tube with the largest internal diameter is desirable to minimize WOB$_{IMP}$. Our data reveal that by removing the inner cannula, WOB$_{IMP}$ is significantly ($p < 0.05$) reduced under all tested conditions. This is most likely due to the resultant increase in the lumen diameter with inner cannula removal. In the patient for whom weaning seems most difficult or unlikely, the reduction in WOB$_{IMP}$ offered by removing the inner cannula and thus increasing the internal diameter may be the edge the patient needs for successful weaning.

This bench study included the typical range of breathing conditions found in patients being weaned from the ventilator. Extraneous variables such as poor pulmonary hygiene, presence of mucus in the tracheostomy tube, and over-hydration via high-flow oxygen delivery devices, are difficult to simulate and, consequently, were not integrated into the lung model.

Conclusions

The imposed WOB was measured in Shiley size 6, 8, and 10 tracheostomy tubes with and without the inner cannula in a lung model. In this study, WOB$_{IMP}$ increased with increases in V_T and frequency, as well as when tube size decreased. WOB$_{IMP}$ decreased significantly in all tubes when the inner cannula was removed, regardless of inspiratory flow demand. In weaning a tracheostomized patient with marginal pulmonary function and reserve, increasing the inner diameter of the tube by removing the inner cannula during spontaneous breathing trials may be beneficial. Clinical studies are needed to determine if these findings are important for the patient.

REFERENCES

1. Heffner JE. Medical indications for tracheotomy. Chest 1989;96(1): 186–190.
2. Hooper M. Nursing care of the patient with a tracheostomy. Nurs Stand 1996;10(34):40–43.
3. Allan D. Patients with an endotracheal tube or tracheostomy. Nurs Times 1984;80(13):36–38.
4. Allan D. Making sense of tracheostomy. Nurs Times 1987;83(45): 36–38.
5. Sugerman HJ, Wolfe L, Pasquale MD, Rogers FB, O'Malley KF, Knudson M, et al. Multicenter, randomized, prospective trial of early tracheostomy. J Trauma 1997;43(5):741–747.
6. Heffner JE, Miller KS, Sahn SA. Tracheostomy in the intensive care unit. Part 1: indications, technique, management. Chest 1986;90(2): 269–274.
7. Maziak DE, Meade MO, Todd TRJ. The timing of tracheotomy: a systematic review. Chest 1998;114(2):605–609.
8. Gunawardana RH. Experience with tracheostomy in medical intensive care patients. Postgrad Med J 1992;68(799):338–341.
9. Banner MJ, Kirby RR, Blanch PB. Site of pressure measurement during spontaneous breathing with continuous positive airway pressure: effect on calculating imposed work of breathing. Crit Care Med 1992;20(4):528–533.
10. Banner MJ, Kirby RR, Blanch PB. Differentiating total work of breathing into its component parts. Chest 1996;109(5):1141–1143.
11. Banner MJ, Kirby RR, Blanch PB, Layon AJ. Decreasing imposed work of the breathing apparatus to zero using pressure-support ventilation. Crit Care Med 1993;21(9):1333–1338.
12. Blanch PB, Banner MJ. Is tracheal pressure ventilator control comparable to an equivalent level of pressure support ventilation? Respir Care 1997;42(11):1022–1033.
13. Banner MJ, Kirby RR, Gabrielli A, Blanch PB, Layon AJ. Partially and totally unloading respiratory muscles based on real-time measurements of work of breathing: a clinical approach. Chest 1994; 106(6):1835–1842.
14. Davis K Jr, Branson RD, Porembka D. A comparison of the imposed work of breathing with endotracheal and tracheostomy tubes in a lung model. Respir Care 1994;39(6):611–616.
15. Bolder PM, Healy TEJ, Bolder AR, Beatty PC, Kay B. The extra work of breathing through adult endotracheal tubes. Anesth Analg 1986;65(8):853–859.
16. Mullins JB, Templer JW, Kong J, Davis WE, Hinson J. Airway resistance and work of breathing in tracheostomy tubes. Laryngoscope 1993;103(12):1367–1372.
17. Marini JJ. Strategies to minimize breathing effort during mechanical ventilation. Crit Care Clin 1990;6(3):635–661.
18. Kirton OC, DeHaven CB, Morgan JP, Windsor J, Civetta JM. Elevated imposed work of breathing masquerading as ventilator weaning intolerance. Chest 1995;108(4):1021–1025.

Electrical Stimulation for Swallowing Disorders Caused by Stroke

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BACKGROUND: An estimated 15 million adults in the United States are affected by dysphagia (difficulty swallowing). Severe dysphagia predisposes to medical complications such as aspiration pneumonia, bronchospasm, dehydration, malnutrition, and asphyxia. These can cause death or increased health care costs from increased severity of illness and prolonged length of stay. Existing modalities for treating dysphagia are generally ineffective, and at best it may take weeks to months to show improvement. One common conventional therapy, application of cold stimulus to the base of the anterior faucial arch, has been reported to be somewhat effective. We describe an alternative treatment consisting of transcutaneous electrical stimulation (ES) applied through electrodes placed on the neck. OBJECTIVE: Compare the effectiveness of ES treatment to thermal-tactile stimulation (TS) treatment in patients with dysphagia caused by stroke and assess the safety of the technique. METHODS: In this controlled study, stroke patients with swallowing disorder were alternately assigned to one of the two treatment groups (TS or ES). Entry criteria included a primary diagnosis of stroke and confirmation of swallowing disorder by modified barium swallow (MBS). TS consisted of touching the base of the anterior faucial arch with a metal probe chilled by immersion in ice. ES was administered with a modified hand-held battery-powered electrical stimulator connected to a pair of electrodes positioned on the neck. Daily treatments of TS or ES lasted 1 hour. Swallow function before and after the treatment regimen was scored from 0 (aspirates own saliva) to 6 (normal swallow) based on substances the patients could swallow during a modified barium swallow. Demographic data were compared with the t test and Fisher exact test. Swallow scores were compared with the Mann-Whitney U test and Wilcoxon signed-rank test. RESULTS: The treatment groups were of similar age and gender ($p > 0.27$), co-morbid conditions ($p = 0.0044$), and initial swallow score ($p = 0.74$). Both treatment groups showed improvement in swallow score, but the final swallow scores were higher in the ES group ($p > 0.0001$). In addition, 98% of ES patients showed some improvement, whereas 27% of TS patients remained at initial swallow score and 11% got worse. These results are based on similar numbers of treatments (average of 5.5 for ES and 6.0 for TS, $p = 0.36$). CONCLUSIONS: ES appears to be a safe and effective treatment for dysphagia due to stroke and results in better swallow function than conventional TS treatment. *Key words: swallowing, dysphagia, electrical stimulation, stroke, modified barium swallow.* [Respir Care 2001;46(5):466–474]

Background

An estimated 15 million adults in the United States[1] are affected by difficulty in swallowing (dysphagia). The prev-alence of dysphagia in certain diseases may approach 90% (eg, amyotrophic lateral sclerosis, Parkinson's disease, and certain types of stroke).[2] Severe dysphagia predisposes to

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medical complications such as aspiration pneumonia, bronchospasm, dehydration, malnutrition, and asphyxia. These can cause death or increased health care costs from increased severity of illness, prolonged length of stay, readmissions, respiratory support, tracheotomy, and percutaneous enterostomal gastric (PEG) tube placement plus related nutritional supplements and equipment.[1-4] Aside from the physical complications of aspiration, patients often suffer severe depression because of the loss of the swallow function and the disruption of normal activities of daily living.

Existing treatments for dysphagia are unable to restore complete swallow function in patients with the most severe disorders. Physical maneuvers to compensate for the deficiency (such as tucking the chin and suck swallow) are considered generally ineffective.[5,6] Thermal-tactile stimulation (TS) (ie, application of cold to the anterior faucial arch[7,8]) and biofeedback[9] have success rates ranging from 0% to 83%.[5,9-11] Studies reporting high success rates with stroke patients generally do not include the most severe forms of dysphagia, in which patients initially aspirate everything, including their own saliva. Often, these studies simply state that improvement was resumption of oral intake, but they do not describe the consistency of the oral intake. The type of oral intake is important because it affects not only hydration and nutrition but also the psychosocial impact on the patient. The minimum goal of treatment should be to achieve sufficient oral intake to prevent or remove a PEG tube, with its attendant difficulties of reflux aspiration and complications associated with infections. The ultimate goal should be restoration of normal swallow.

Current modalities have long treatment times: 2–52 weeks (average 15 weeks) for severe dysphagia using tactile and thermal-tactile stimulation[5] and 3–29 weeks using biofeedback.[9] A 4-fold increase in pneumonia has been documented during treatment, compared to the post-treatment period.[5] Lengthy treatment of swallowing disorders is thus risky and may potentially interfere with treatment of other medical problems.

Spontaneous improvement in swallowing may occur in certain acute diseases that cause mild dysphagia.[12] However, in the United States only 2% of patients with neurologic disorders and PEGs returned to full oral feeding after one year, suggesting that spontaneous improvement is rare for cases of severe dysphagia.[3]

Electrical stimulation (ES) has been reported as a treatment for dysphagia.[13,14] Park et al[15] applied electricity through a prosthetic device on the soft palate, aiming to re-educate neural pathways associated with the swallowing reflex. They reported a 50% success rate in improving the swallow of patients already capable of oral feeding. Transcutaneous application of electrical current to the neck with a nerve stimulator has also been successful in improving swallow function, but has rarely been used, because of assumed concerns for safety.[6,16]

We report a new treatment for dysphagia, consisting of transcutaneous ES applied through electrodes placed on the neck. The purpose of this study was to compare the effectiveness of ES to TS in patients with dysphagia caused by stroke, and to assess the safety of the technique. Because ES is a more direct stimulus than TS to nerves and muscles associated with swallowing, we hypothesized that ES would result in better swallow function than TS in patients with comparable conditions of dysphagia. We also monitored patients after treatment to investigate the long-term effects of treatment and the potential for spontaneous recovery.

Methods

The study was conducted at Hillcrest Hospital, a 280-bed acute care hospital in a suburb of Cleveland, Ohio. All new referrals who met entry criteria and signed the consent form were enrolled during the study period. The study period was September 23, 1993, through January 24, 1995. The study population included both in-patients and out-patients. Entry criteria included:
- primary diagnosis of stroke
- confirmation of swallowing disorder by modified barium swallow (MBS)

Exclusion criteria were:
- inability to complete at least 2 consecutive days of therapy
- any behavioral disorder that interfered with administration of therapy
- substantial reflux from feeding tube
- dysphagia from drug toxicity

Duration of swallow dysfunction did not limit eligibility. Written, informed consent, as approved by the institutional review board, was obtained from all patients.

Stroke patients with possible swallowing disorder were alternately assigned to one of the 2 treatment groups (TS or ES) independent of any other information and before being seen by the speech-language pathologist. After assignment, the speech-language pathologist performed the MBS with a radiologist to determine the severity of the swallowing disorder and to assign a swallow score (see assessment protocol below). If it was confirmed that the patient did not meet any exclusion criteria, the treatment regimen was begun. No patients were excluded from the study because of the severity of dysphagia. After the course of treatment, another MBS was performed and a final swallow score assessed.

Assessment Protocol

Each patient's swallow function was evaluated via standardized MBS,[8,17] with the addition of following the bolus

into the stomach to identify esophageal reflux that could result in aspiration. Patients swallowed various consistencies of food mixed with barium powder while being observed under fluoroscopy. Food consistencies progressed from thick to thin, until aspiration occurred. Penetration was defined as entry of the bolus into the laryngeal vestibule. Aspiration was defined as passage of barium below the level of the vocal cords. The results of the MBS were interpreted as a swallow score according to the criteria listed in Table 1.

The swallow score was assigned as follows. The speech therapist would perform the MBS and send the videotape of the procedure to a designated radiologist. The radiologist would then provide a narrative interpretation of the tape in terms of what type of liquid could be safely swallowed. That narrative report was sent back to the speech therapist, who then assigned the corresponding score (Table 1). There were 3 radiologists who assigned scores, and at the time of scoring they did not know which treatment a patient had received.

The MBS procedure we used was standard except for 2 items. First, instead of barium paste, we used barium powder, because it has less effect on the consistency and taste of the liquid it is mixed with. The idea is to create mixtures of different, realistic consistencies but with as much of the original taste as possible. Paste has a greater tendency to thicken the mixture than powder and also has a more objectionable taste. The second difference was in the order of consistencies presented to the patient. Standard references suggest using thin liquid (eg, water), then pudding, and then cookie.[18] The problem with this order is that thin liquids may be (but are not always) the most easily aspirated.[18] Thus, if the patient aspirates early in the procedure because thin liquid was used first, then (a) the airway becomes contaminated with barium, making visualization of aspiration for other substances difficult, and (b) because of the aspiration, the procedure may be terminated without determining what consistency can be safely swallowed.

During treatment, the speech-language pathologist auscultated the right main bronchus during inspiration. A normal swallow was a single or polysyllabic sound of 1–2 seconds duration, representing the movement of food through the pharyngeal area and into the esophagus, and consisted of only clear breath sounds.[19] This technique enabled the therapist to identify abnormal swallowing or so-called silent aspiration by airway sounds, including rales and rhonchi, during post-swallow inspiration. Silent aspiration is a condition in which food or liquid enters the airway but does not produce any obvious signs of aspiration (ie, there is no cough during or after the swallow).[20] The use of auscultation of the right bronchus during inspiration and following ingestion of the food or liquid bolus aided in hearing changes in lung sounds and changes in the rate of respiration, which often trigger concern about silent aspiration and justify an MBS. Swallow function (by auscultation) was assessed each day of treatment protocol to check for silent aspiration.

Treatment Protocols

General Treatment Protocol. In-patient treatment (either ES or TS) began within 24 hours of initial evaluation. Duration was 1 hour per day of treatment and 10 minutes of challenge/assessment. If a patient became fatigued, treatment was continued later in the day, as often as necessary, to obtain the full hour. Treatment continued on consecutive days until a swallow function score of at least 5 was achieved or the patient was discharged because of insurance constraints. Those patients discharged before achieving a score of 5 avoided a PEG if they could achieve a score of at least 2 on consistency of liquid.

Out-patients were treated 3 times per week for 1 hour per treatment. Treatment continued until they achieved a swallow score of 6 or it was judged that no more progress would be made.

Follow-up on patients was based on medical records (for readmission) or consultation with the patient, family, physician, or nursing home therapists, for up to 3 years.

Table 1   Swallow Function Scoring System*

Swallow Function Score	Safe Liquid Consistency	Clinical Implication	Level of Swallow Deficit
0	Nothing safe (aspirates saliva)	No solid or liquid is safe	Profound
1	Saliva	Same as above (candidate for PEG)	Profound
2	Pudding, paste, ice slush	—	Substantial
3	Honey consistency (liquid with thickener or premixed product like *Resource* brand liquid nourishment)	—	Moderate
4	Nectar consistency (pureed fruit juice such as apricot, peach, pear)	—	Mild
5	Thin liquids (eg, cream soups, orange juice, carbonated beverage)	No coffee, tea, thin juice (eg, apple), or water	Minimal
6	Water	All liquids tolerated	Normal

*This system identifies the consistency of liquid that the patient can swallow without aspiration
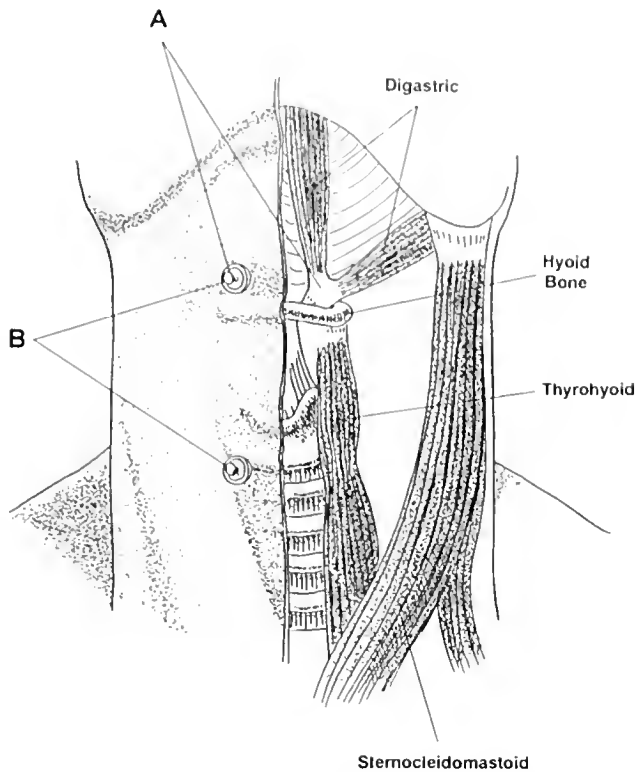
Fig. 1. Diagram of the throat showing placements for pairs of snap electrodes. One of two placements was used: (A) On either side of the midline, above the lesser horns of the hyoid bone, on the digastric muscle. (B) On either side of the midline (preferably on right side) with upper electrode above lesser horns of the hyoid bone, on the digastric muscle, and lower electrode on the thyrohyoid muscle at the level of the top of the cricothyroid cartilage. Position A was used for patients with tracheostomies or those whose anatomy prevented using the other position. Position B was used for everyone else.

Thermal-Tactile Stimulation Treatment Protocol. TS was given in three 20-minute intervals daily. A speech pathologist (one of the authors, MLF) used the standard methodology[8] for TS, including verbal coaching. TS was applied with a size 00 oral examination mirror cooled by immersion in ice. The base of the anterior faucial arch was lightly touched with the mirror back. The mirror was removed, and the patient was asked to close his or her mouth and attempt to swallow saliva (dry swallow). TS and verbal coaching continued. If a dry swallow was elicited, the patient was challenged with thickened liquids (pudding viscosity).

Electrical Stimulation Treatment Protocol. ES was administered by a physical therapist in conjunction with a speech pathologist (MLF), using a modified hand-held battery-powered electrical stimulator (Staodyn EMS + 2, Staodyn Inc, Longmont, Colorado). Electrodes were placed on the neck in one of two positions (Fig. 1) and were repositioned until muscle fasciculations occurred or the stron-

gest contraction was observed during the swallow response. Neuromuscular ES consisted of a symmetric rectangular alternating current passing between positive and negative snap skin electrodes. Frequency and pulse width were fixed at 80 Hz and 300 microseconds. Current intensity was set to the patient's tolerance and comfort level. Tolerance and comfort differed among individuals. The sensation most patients experienced first was a very slight tingling or crawling sensation. As the intensity was increased (in 2.5 mA increments from a start of 2.5 mA up to a maximum of 25.0 mA), the individual perceived a strong vibration or the sensation that the electrodes were coming loose from the neck. Most individuals accommodated rapidly enough to the sensations that the intensity could be continuously increased until contractions were consistently audible (designated the therapy current level). When ES was successful in obtaining a voluntary swallow response, the patient was asked to attempt a swallow with a specific oral consistency. ES was delivered at the therapy current for a total of 60 minutes per treatment, in the continuous mode, with a 1.0 second pause between each minute.

All patients were monitored continuously by electrocardiography and pulse oximetry. A pulse oximetry-measured blood oxygen saturation (S_{pO_2}) decrease of more than 2% was considered a desaturation due to aspiration. Laryngospasm was defined as a spasmodic closure of the glottis with severely limited ability to ventilate. Laryngospasm was judged by the speech therapist, during treatment, based on audible or visible signs of respiratory distress. All recordings were reviewed and interpreted by the medical chief of staff of the acute care facility.

Data Analysis

Unpaired t tests were used to compare the mean ages and the total number of treatments in the two groups. The Fisher exact test was used to compare the proportions of females to males in each group. The similarity of co-morbid conditions was evaluated with Kendall's tau test (ie, if a high proportion of TS patients have a co-morbid condition, do a high proportion of ES patients also have the co-morbid condition, and vice versa). The proportions of confounding factors (ie, brainstem vs hemispheric vs multiple strokes) in the 2 groups were compared with the chi-square test. The Mann-Whitney U test was used to compare the initial swallow scores (ie, to determine if the initial degree of dysphagia on entering the study was the same for both groups) and the distributions of final swallow scores (ie, to determine if one treatment group showed greater improvement). The change in swallow scores (ie, initial vs final) was evaluated with the Wilcoxon signed-rank test. Analyses were performed with StatView software (SAS Institute Inc, Cary, North Carolina). Statistical significance was set at $p < 0.05$.

Results

One hundred twenty-five patients were screened for possible inclusion in the study. Fifteen refused to sign consent after meeting entry criteria, leaving 110 who were enrolled. Ninety-nine patients completed the study (Table 2). All TS patients were in-patients. All but 6 ES patients were in-patients, and one was both an in-patient and out-patient. Eleven patients dropped out of the study: 6 had drug toxicity from other treatments, 2 were transferred to other hospitals, and 3 dropped out for unrecorded reasons.

The 2 treatment groups were comparable in terms of mean age and gender distribution and in co-morbid conditions that would affect treatment outcome (see Table 2). The condition that would most negatively affect the conventional treatment group was dementia, and the prevalence was identical in the 2 groups. The presence of confounding factors related to the type of lesion (ie, brainstem vs hemispheric stroke vs multiple strokes) was similar in both groups (Table 3). The TS and ES treatment groups had similar distributions of initial swallow score (p = 0.74). There were aphasic patients in both groups, but aphasia did not affect their treatment. There were no patients in the study with apraxia of swallowing. There were 7 ES versus 6 TS patients with dysarthria, but in no case did dysarthria appear to affect outcome.

Both treatment groups showed improvement in swallow score (Table 4). However, Figure 2 shows that ES resulted

Table 2. Treatment Groups with Respect to Demography and Health

Variable	Thermal Stimulation (n = 36)	Electrical Stimulation (n = 63)	p
Average age	78.1	75.7	0.27
Maximum age	91	101	—
Minimum age	65	49	—
Female (%)	44	48	0.83
Co-morbid conditions†	(%)	(%)	
Stroke	8	11	
Coronary artery disease	8	8	
Congestive heart failure	14	8	
Chronic obstructive pulmonary disease	6	5	
Hypertension	17	19	
Dementia	3	3	
Diabetes mellitus	6	8	
Parkinson's disease	0	2	
Cancer	25	10	
Multiple sclerosis	3	0	

† Patients often had more than one co-morbid condition; ie, proportions were not mutually exclusive, and proportions were significantly correlated by Kendall's tau (p = 0.0044)

Table 3. Frequencies of Types of Lesions*

Treatment	Brainstem	Hemispheric	Multiple Strokes
Electrical stimulation	7	29	24
Thermal-tactile stimulation	1	19	8

*Not all patients were evaluated for type of lesion. The proportion of observations in the different categories is not significantly different than would be expected from random occurrence (p = 0.183)

in more people having higher final swallow scores than TS (p < 0.0001). In addition, all but one of the ES patients showed some improvement (98%; the one patient remained at a swallow score of 2), whereas 17 (27%) of TS patients remained at initial swallow score and 4 (11%) got worse.

ES patients with +6 changes progressed from swallow function 0 (completely dysphagic) to swallow function 6 (normal swallow). ES patients with +5 changes included 3 patients who progressed from swallow function 1 (tolerates saliva only) to 6, and 6 patients who progressed from swallow function 0 to swallow function 5. Other step changes less than +5 include some ES patients who achieved swallow functions 5 or 6, but these patients started with swallow function greater than 1. No TS patient, regardless of initial swallow function, achieved a final swallow function greater than 4. These results are based on similar numbers of treatments (average of 5.5 for ES and 6.0 for TS, p = 0.36).

Several focused comparisons illustrate further differences between ES and TS. For patients starting at swallow scores 0 and 1, achieving swallow score 2 or higher indicated successful treatment, in that PEG was not required. Only 52% (15 of 29) of TS patients experienced successful treatment, compared to 95% (41 of 43) of ES patients (p < 0.0001). ES treatments were also more successful than TS treatments, based on achievement of complete swallow score 6 (35% of ES patients vs 0% of TS patients, p < 0.0002), each starting at swallow score 0 or 1. In addition, 4 TS patients (11%) required a PEG during treatment. None of the 58 ES patients required a PEG during treatment, and a swallow score of 2 was achieved within 1–2 treatments in all ES patients.

Twenty-five bedside evaluations performed by the therapist (ie, auscultation of the right bronchial tree for evi-

Table 4. Mean Swallow Scores Before and After Treatment

Treatment	Initial Swallow Score	Final Swallow Score
Electrical stimulation	0.76 ± 1.04	4.52 ± 1.69
Thermal-tactile stimulation	0.75 ± 1.20	1.39 ± 1.13

Values are ± standard deviation

Electrical Stimulation
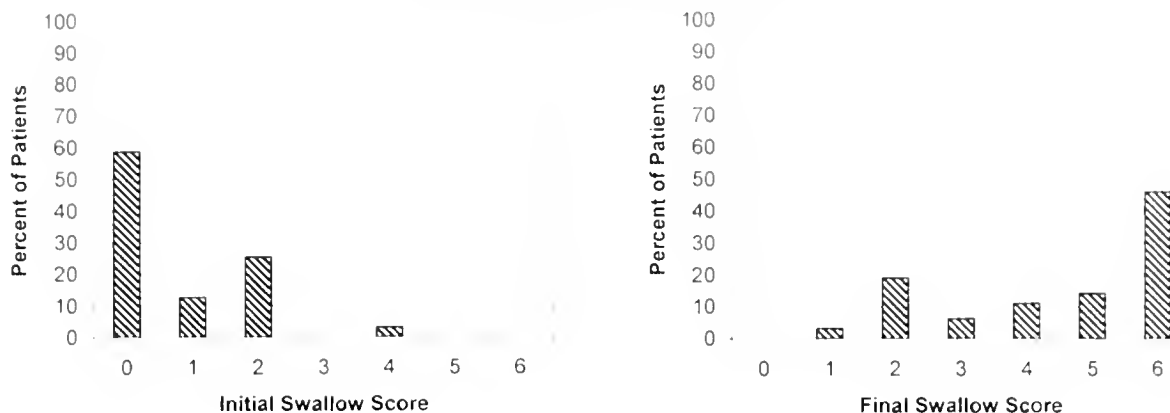
Thermal-Tactile Stimulation
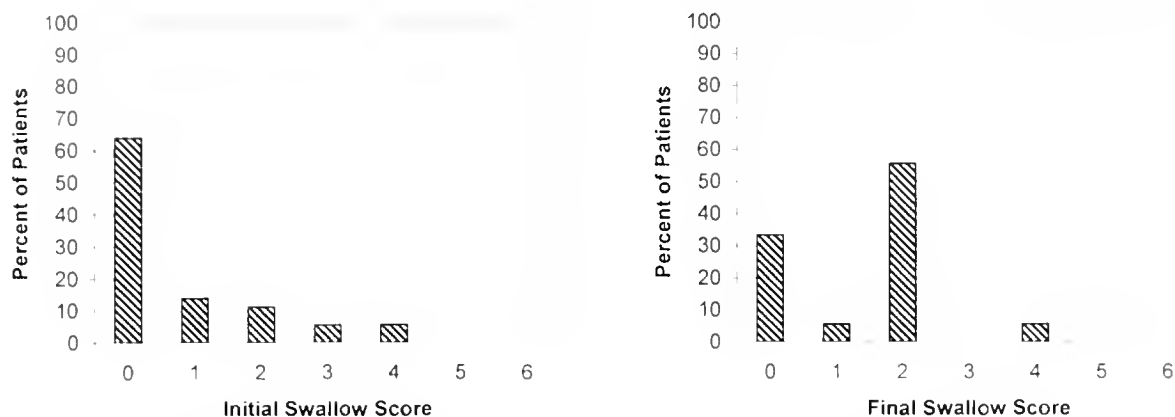
Fig. 2. Distributions of initial and final swallow scores for electrical stimulation and thermal-tactile stimulation treatment groups. A higher score means better swallow function. Initial swallow scores for the two groups were similar ($p = 0.74$). Both groups showed improvement in score (thermal-tactile stimulation $p = 0.0048$; electrical stimulation $p < 0.0001$). The electrical stimulation group had higher final scores ($p < 0.0001$).

dence of rhonchi or change in ventilatory pattern) were compared with corresponding MBS studies interpreted by a radiologist. Only one of the 25 comparisons disagreed: the therapist judged silent aspiration that was not confirmed by MBS. This yields the decision matrix shown in

Table 5. Analysis of Agreement between Bedside Assessment of Silent Aspiration and Results of Modified Barium Swallow

Bedside Evaluation	MBS Interpretation	
	Aspiration Present	Aspiration Absent
Aspiration present	24	1
Aspiration absent	0	0

MBS = modified barium swallow

Table 6. Proportions of Patients in Post-Treatment Categories

Category	Thermal Stimulation (n = 33)	Electrical Stimulation (n = 52)
No change for >2 y, alive	0.061	0.289
No change for >2 y, lost	0.242	0.269
No change for >2 y, died	0.364	0.250
Improved within 2 y	0.000	0.077
Aspiration or PEG	0.242	0.000
New episode of dysphagia	—	0.115
Received ES after TS	0.091	

* Average time of follow up = 1 year
Proportion is 0.143 of 28 electrical stimulation patients with final swallow function = 7
PEG = percutaneous enterostomal gastric tube
ES = electrical stimulation
TS = thermal-tactile stimulation
Full swallow function restored after electrical stimulation

Table 5. The positive predictive value was 24/25 = 96%; the true positive rate was 24/24 = 100%; the false positive rate was 1/25 = 4%.

Follow-up data show that the effects of treatments administered during the study generally persisted (Table 6). Most patients retained their final swallow function for over 2 years (89% for ES and 67% for TS). Loss of swallow function during the post-treatment period for ES patients was based on new episodes of the problems that caused the dysphagia. None of the TS patients showed improved swallow function, whereas 4 (14%) of ES patients improved (3 confirmed by MBS). There was a high rate of aspiration (24%) in TS patients, compared with no aspiration in ES patients. Two of the aspirating TS patients received a PEG.

A total of 318 applications of ES were administered to patients during this study. Not a single case of laryngospasm or decrease in S_{pO_2} was observed. No change in heart rhythm occurred, based on electrocardiograph rhythm strip recordings.

Discussion

The demographic similarities between the two groups (Table 2) indicate that the desired properties of randomization from the same underlying population were in fact achieved for the two treatment groups, despite the fact that a strict randomization scheme was not used.[21] There was, however, one general difference between the two groups: the ES group was treated much longer after stroke than the TS group. This is because most of these patients had already failed conventional therapy, which was the reason they were referred for the study. The longer the period after the stroke, the less success is expected with dysphagia treatment. Despite this potential bias against the ES treatment, the ES group showed better results than the TS group.

Bedside evaluations are important in determining the safety of treatment, to estimate the patient's progress during the treatment period, and to justify further MBS studies. In our study, auscultation was used to detect silent aspiration during treatment. The ability to detect aspiration by this method was evaluated by comparison with radiographic evidence of aspiration. However, MBS procedures were done only for patients who were suspected of aspiration (silent or not). Therefore, we collected no data from which negative predictive value could be estimated, yet the high positive predictive value suggests that auscultation deserves further study as a potentially useful screening test for silent aspiration. More research should be done to identify the optimum bedside evaluation technique and to compare its accuracy with the gold standard, MBS.

Application of ES to muscles associated with swallowing links swallowing therapy with physical therapy. A fundamental principle of physical therapy is that disuse of a striated muscle leads to atrophy of that muscle, even if the medical condition leading to disuse has no direct effect on the muscle or associated nerves.[22] Loss of muscle tone is identified by physical therapists as little or no measurable contractility or strength. When attempts at exercise alone fail to result in contraction of an atrophied muscle, ES may enhance tone to the point where exercise may strengthen or activate the muscle.

There may be an analogy with dysphagia. A medical event such as stroke may block the primary neural pathway for swallowing. There are fewer myofibrils per motor unit of the laryngeal muscles relative to larger muscles (4–6 vs 4,000), and there are numerous small muscles of this type that participate in the oropharyngeal phase of swallow.[23] In addition, the motor units within each laryngeal muscle tend to fire asynchronously during a normal swallow, contrasting with the more synchronous firing of larger muscles designed for strength.[23] Under this model, even a few days without the typical 600–2,400 normal swallows per day[24,25] could lead to long-term dysphagia. Though this design of small muscles might make them more susceptible to failure from lack of use, it is possible that this design can respond more fully to ES. Perhaps this is a reason why ES of the neck restores effective swallow with fewer treatments than required for restoration of appropriate function by ES of other muscles of the body.[26] Alternatively, fewer treatments might be associated with stimulating a reflex, since swallowing is a complex action that is usually initiated voluntarily but is always completed as a reflex involving afferent and efferent cranial nerves[27,28] and primary and secondary swallow centers in the cortex.[29] These muscle tone and reflex hypotheses also pertain to the success of ES in treating urinary incontinence.[30] Much research is required to determine whether ES, applied at a sensory level in our study, works via a peripheral nerve, a direct effect on the small muscles, the central nervous system, or a combination of these factors.

Our data directly address issues of safety. ES of the head and neck, discussed in the recent third edition of Charles Darwin's *The Expression of the Emotions in Man and Animals*,[31] has been the subject of major recent debate about safety. Possible risks include arrhythmia, hypotension, interference with pacemaker, laryngospasm, glottic closure, burns, and tumor growth.[27] However, one successful study that applied external ES to a nerve of the neck had no complications.[16] Other studies also observed no change in vital signs, electrocardiograph, or other adverse effects in patients who received implantable recurrent laryngeal and vagal nerve stimulators used to treat spastic dysphonia or control epilepsy.[27,28] External application of ES with a muscle stimulator within the settings used in our study appears safe, at the sensory level of application. Standard electrode placement in our study purposely avoids the carotid body. In addition, the voltage

and current used in our device are lower than is delivered by a standard neuromuscular stimulator, assumed by other authors concerned over the safety of ES.

The most important theoretical risk of ES is laryngospasm. In an animal study, laryngospasm was achieved with repetitive suprathreshold ES, but not with single-shock excitation of the superior laryngeal nerve.[27] As stimulus frequency went above 32–64 Hz, there was a decrease in adductor after-discharge and glottic pressure.[28] In our study, suprathreshold levels of stimulation of the superior laryngeal nerve did not occur, because of the level of therapeutic current, limits on the maximum current of the stimulator, and attenuation by soft tissues of the neck. The high-frequency stimulation of ES for dysphagia exceeded 64 Hz and may be one of the factors protecting against laryngospasm. In addition, the constant current stimulator automatically dropped the voltage to maintain a constant current dose in the event of decreased electrode or tissue resistance. With these safeguards, a device as configured for our study is apparently safe. The hypothetical concerns about safety are not supported by our data.

Although there are reports in the literature that stroke patients can recover their swallow spontaneously,[12] tube feedings were needed for 15–60 weeks.[5,9,10] Howard et al[3] indicated that 30% of all patients continued on total tube feeding at one year after stroke. The patients who received ES in our study began eating following 3 treatments and did not require tube feeding thereafter. ES may initiate muscle reeducation prior to the beginning of spontaneous recovery and prevent the need for tube feeding.

In an age when extensive efforts are made to reduce health care costs, the ES protocol can contribute substantially to those efforts. Between 300,000 and 600,000 new cases of dysphagia occur each year in stroke patients.[21] In 1992, the cost of United States enteral nutrition in neurologic disease alone exceeded 330 million dollars per year.[3] Since the ES protocol restored swallow function to a score of 2 within 1–2 days of treatment, a hospitalized stroke patient who lost swallow function in association with the underlying medical problem could eat on his or her own or with reduced assistance as an in-patient. Six treatments of one hour each day, for in-patients or out-patients, would be expected to restore normal swallow in 35% of the most severe cases of stroke and 45% of all stroke cases. The medical implications for patients include reduced amounts of therapy (fewer sessions, less traveling), avoidance of surgery for PEG (and attendant complications), avoidance of specialized dietary regimens, normal liquid intake, and reduced risk of aspiration pneumonia. Caregivers benefit from increased efficiency. Corrected dysphagia would interfere less with treatments for other medical problems while improving cost effectiveness for health care facilities. The social implication of lower medical bills and less restricted social activities associated with eating is higher quality of life for both the patient and the family.

A potential limitation of this study is that, though the scoring of swallow function was fairly objective (see Table 1), it does not preclude subjective bias. However, we compared the distribution of final swallow scores of 29 TS patients from our study with that of 53 patients treated with TS by Neumann et al[5] and found no difference (Kolmogorov-Smirnoff test KS = 0.2531, p = 0.13). Therefore the difference between TS and ES was probably not due to bias against TS. The physical evidence of MBS reveals no bias in favor of ES. In addition, the swallow function score we used is no more subjective than the score validated and published by Rosenbek et al.[7] The major difference with our score is that we did not record the trajectory of the bolus, but only whether it was aspirated and the consistency of liquid aspirated. Because consistency affects risk of aspiration, the purpose of the score is to rank the consistency of liquid that can be safely swallowed. This is the type of information referring physicians prefer to see as an interpretation of the MBS procedure because it helps them formulate instructions for the patient.

Conclusions

Transcutaneous ES appears to be a safe and effective treatment for dysphagia caused by stroke, and it results in better improvement in swallow function than does thermal-tactile stimulation. Normal swallow function was restored to 35% of the most severely dysphagic patients in less than a week of daily treatment, to 45% of patients at all levels of severity, and the restoration persisted until a new episode of dysphagia occurred. The only limitations of ES are that it cannot be done on patients who talk continuously (such as is found in some severely demented patients), patients who have beards must be willing to shave them for ES, and TS treatment requires the patient's cooperation in opening the mouth and in following verbal commands.

REFERENCES

1. Bello J, editor. Prevalence of speech, voice, and language disorders in the United States. Communication Facts. 1994 ed. American Speech-Language-Hearing Association 1-4.
2. Gordon C, Hewer RL, Wade DT. Dysphagia in acute stroke. BMJ 1987;295(6595):411-414.

3. Howard L, Ament M, Fleming CR, Shike M, Steiger E. Current use and clinical outcome of home parenteral and enteral nutrition therapies in the United States. Gastroenterology 1995;109(2):355-365.

4. Hogue CW, Lappas GD, Creswell LL, Ferguson TB, Sample M, Pugh D, et al. Swallowing dysfunction after cardiac operations: associated adverse outcomes and risk factors including intraoperative transesophageal echocardiography. J Thorac Cardiovasc Surg 1995; 110(2):517-522.

5. Neumann S, Bartolome G, Buchholz D, Prosiegel M. Swallowing therapy of neurologic patients: correlation of outcome with pre-treatment variables and therapeutic methods. Dysphagia 1995;10(1): 1-5.

6. Langmore S, Miller RM. Behavioral treatment for adults with oro-pharyngeal dysphagia. Arch Phys Med Rehabil 1994;75(10):1154-1159.

7. Logemann J. Evaluation and treatment of swallowing disorders. San Diego: College Hill Press; 1983: 134-223.

8. Lazzara G, Lazarus C, Logemann JA. Impact of thermal stimulation on the triggering of the swallowing reflex. Dysphagia 1986;1(2):73-77.

9. Crary MA. A direct intervention program for chronic neurogenic dysphagia secondary to brainstem stroke. Dysphagia 1995;10(1):6-18.

10. Rosenbeck JC, Robbins J, Fishback B, Levine RL. Effects of thermal application on dysphagia after stroke. J Speech Hear Res 1991;34(6): 1257-1268.

11. Singh V, Brockbank MJ, Frost RA, Tyler S. Multidisciplinary management of dysphagia: the first 100 cases. J Laryngol Otol 1995; 109(5):419-424.

12. Barer DH. The natural history and functional consequences of dysphagia after hemispheric stroke. J Neurol Neurosurg Psychiatry 1989; 52(2):236-241.

13. Freed M, Christian MO, Beytas EM, Tucker H, Kotton B. Electrical stimulation of the neck: A new effective treatment for dysphagia (abstract). Dysphagia 1996;11:159.

14. Chatburn RL, Freed M. Electrical stimulation for treatment of dysphagia in children failing conventional therapy (abstract). Respir Care 2000;45(8):1009.

15. Park CL, O'Neill PA, Martin DF. A pilot exploratory study of oral electrical stimulation on swallow function following stroke: an innovative technique. Dysphagia 1997;12(3):161-166.

16. Larsen GL. Conservative management for incomplete dysphagia paralytica. Arch Phys Med Rehabil 1973;54(4):180-185.

17. Ott DJ, Pikna LA. Clinical and videofluoroscopic evaluation of swallowing disorders. AJR Am J Roentgenol 1993;161(3):507-513.

18. Logemann JA. Evaluation and treatment of swallowing disorders. Austin: Pro-Ed, 1998: 177.

19. Leonard R, Kendall K, editors. Dysphagia assessment and treatment planning. San Diego: Singular Publishing Group, 1997.

20. Carrau RL, Murray T. Comprehensive management of swallowing disorders. San Diego: Singular Publishing Group; 1999: 72.

21. Fisher RA. The design of experiments. Edinburgh: Oliver & Boyd, 1935: 13-29.

22. Gordon T, Mao J. Muscle atrophy and procedures for training after spinal cord injury. Phys Ther 1994;74(1):50-60.

23. West JB. Best and Taylor's physiological basis of medical practice, 12th ed. Baltimore: Williams & Wilkins, 1991.

24. Diagnosis and treatment of swallowing disorders (Dysphagia) in acute-care stroke patients. Prepared for: Agency for Health Care Policy and Research (U.S. Department of Health and Human Services). AHCPR Publication No. 99-E024. July 1999. available at http://www.ahcpr.gov/clinic/dysphsum.htm

25. Rosenbek JC, Robbins JA, Roecker EB, Coyle JL, Wood JL. A penetration-aspiration scale. Dysphagia 1996;11(2):93-98.

26. McQuain MT, Sinaki M, Shibley LD, Wahner HW, Ilstrup DM. Effect of electrical stimulation on lumbar paraspinal muscles. Spine 1993;18(13):1787-1792.

27. Friedman M, Wernicke JF, Caldarelli DD. Safety and tolerability of the implantable recurrent laryngeal nerve stimulator. Laryngoscope 1994;104(10):1240-1244.

28. Sasaki CT, Suzuki M. Laryngeal spasm: a neurophysiologic redefinition. Ann Otol Rhinol Laryngol 1977;86(2 Pt 1):150-157.

29. Hamdy S, Aziz Q, Rothwell JC, Singh KD, Barlow J, Hughes DG, et al. The cortical topography of human swallowing musculature in health and disease. Nat Med 1996;2(11):1217-1224.

30. Fall M, Lindstrom S. Electrical stimulation: a physiologic approach to the treatment of urinary incontinence. Urol Clin North Am 1991; 18(2):393-407.

31. Darwin C. The expression of the emotions in man and animals. 3rd ed. New York: Oxford University Press; 1998: 21: 278-309.

Partnering for Optimal Respiratory Home Care: Physicians Working with Respiratory Therapists to Optimally Meet Respiratory Home Care Needs

Greg Spratt RRT CPFT and Thomas L Petty MD FAARC

Introduction
What Is the Need for Respiratory Home Care?
   Conditions Requiring Respiratory Home Care
   Influences on the Need for Respiratory Home Care
How Is Optimal Respiratory Home Care Delivered?
   Team Approach
   Respiratory Therapists in the Home
   Benefits of Respiratory Home Care
How Does Reimbursement Affect the Quality of Care?
   Influence of Equipment Reimbursement Methods on Quality of Care
   Effect of Recent Cuts
Discussion
Summary

The need for respiratory care services continues to increase, reimbursement for those services has decreased, and cost-containment measures have increased the frequency of home health care. Respiratory therapists are well qualified to provide home respiratory care, reduce misallocation of respiratory services, assess patient respiratory status, identify problems and needs, evaluate the effect of the home setting, educate the patient on proper equipment use, monitor patient response to and complications of therapy, monitor equipment functioning, monitor for appropriate infection control procedures, make recommendations for changes to therapy regimen, and adjust therapy under the direction of the physician. Teamwork benefits all parties and offers cost and time savings, improved data collection and communication, higher job satisfaction, and better patient monitoring, education, and quality of life. Respiratory therapists are positioned to optimize treatment efficacy, maximize patient compliance, and minimize hospitalizations among patients receiving respiratory home care. *Key words: respiratory home care, respiratory therapist, reimbursement, allocation.* [Respir Care 2001;46(5):475–488]

Introduction

The American Association for Respiratory Care (AARC) has defined "respiratory home care" as

... those forms of respiratory care provided in the patient's place of residence by personnel trained in respiratory care working under medical supervision. The goals of respiratory home care are to improve the patient's physical well being and potential for productivity, and to promote self-sufficiency within the individual's limitations.[1]

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Respiratory services commonly provided to the home setting include: equipment, accessories, and supplies used for therapeutic interventions; assessment, diagnostic, and monitoring procedures; and respiratory therapists (RTs) to provide patient education, rehabilitation services, disease and case management, and to conduct research (Table 1).

RTs have been recognized by many physician organizations as the most appropriate ancillary health care personnel to provide home respiratory services.[2-5] The National Association for the Medical Direction of Respiratory Care (NAMDRC) statement reads, "NAMDRC unequivocally supports the premise that RTs are the nonphysician care givers who are best qualified by both education and examination to render respiratory care services in the hospital and alternate sites, including the home."[3] The California Thoracic Society states, "The RCP (respiratory care practitioner) is qualified to assist the physician in assessing the overall needs of patients, and recommending and delivering necessary care."[5]

What Is the Need for Respiratory Home Care?

Conditions Requiring Respiratory Home Care

An estimated 20% of patients over age 65 have functional impairments with related home care needs that are often unrecognized during the typical office visit.[6] Diseases requiring the provision of quality home respiratory services are on the increase. Chronic obstructive pulmonary disease (COPD) is currently the fourth leading cause of death in the United States, and its incidence has increased 41.5% since 1982. Sixteen million Americans with COPD have been *identified*, and it is estimated that 30–35 million Americans may be afflicted. COPD is the only major cause of death in which the numbers are rising. While mortality from heart disease (number one cause of death) decreased 45% and cardiovascular disease mortality (number two cause) decreased 58%, mortality from COPD increased 32.9% from 1979 to 1991. Direct costs for the care of COPD are estimated at $7–15 billion annually.[7] In 1998, the National Heart, Lung, and Blood Institute placed the total annual cost of COPD in America at $26 billion.[8] Some are predicting that unless current trends are reversed, COPD is likely to be the biggest health problem of the new millennium.[9] At least two major efforts are directed at addressing these growing trends. The National Lung Health Education Program is a national effort endorsed by several major physician organizations, the AARC, and the National Heart Lung and Blood Institute. An international effort, the Global Obstructive Lung Disease Initiative has been founded by the World Health Organization to explore this problem from an international perspective.

Asthma, another disease frequently requiring respiratory home care, presents a similar picture. Asthma affects 14–15 million Americans, and the incidence of asthma is also increasing.[10] It is the most common chronic disease of

Table 1   Respiratory Services Currently Provided in the Home Setting

Therapeutic
  Aerosol therapy
    Bland aerosol
    Hand-held (jet) nebulizer
    Ultrasonic nebulizer
    Continuous aerosol
    Croup tents
    Inhaled medications
      Beta adrenergic agonists
      Anticholinergics
      Mediator blocking agents
      Steroids
      Pentamidine
      Other medications
  Oxygen therapy
    High pressure cylinders
    Oxygen concentrators
    Liquid oxygen
    Transtracheal oxygen
    Portable oxygen
      Portable liquid
      Portable high-pressure cylinders
      Oxygen conservation devices
  Ventilation
    Invasive positive pressure ventilation
    Noninvasive positive pressure ventilation
    Negative pressure ventilation
    Abdominal belts, rocking beds, and other hybrid ventilation devices
  Lung expansion therapy
    Incentive spirometry
    Intermittent positive pressure breathing
  Metered-dose inhalers and chambers/spacers
Diagnostics and monitoring
  Spirometry
  Peak flow
  Oximetry recording
  Sleep studies/polysomnography
  End-tidal carbon dioxide/capnography
  Cardiac event monitors
  Infant cardiopulmonary monitors/event recorders
Patient and home assessment
  Physical assessment
  Home environmental assessment
  Response to therapy
Patient education
Rehabilitation services
  Activity/exercise programs
Disease/case management
Research

childhood, affecting 7.4% of children ages 5–14 years and 4.8 million children under 18 years of age. Asthma prevalence in preschool children was estimated at 5.8% of children under age 5 in 1994 (as reported by a family member), a 160% increase since 1980.[11] In 1994, 5.4% of Americans reported having asthma, a 75% increase since 1980. There are more than 5,000 asthma deaths[12] and 470,000 asthma hospitalizations annually, and there were 1.9 million emergency room visits for asthma in 1995. Health care costs are estimated at more than $6 billion annually,[13] with another $1 billion in lost productivity. Asthma resulted in 100 million days of restricted activity and more than 10 million missed school days.[14]

Sleep apnea and pneumonias associated with human immunodeficiency virus/acquired immunodeficiency syndrome are also on the increase.[15-17] Other cardiopulmonary diseases such as congestive heart failure, stroke, and lung cancer frequently require home respiratory services. Even patients with nonpulmonary diagnoses may require home respiratory services. This is especially true in neuromuscular diseases such as motor neuron disease, muscular dystrophy/atrophy, spinal cord injury, myasthenia gravis, and diaphragmatic paralysis, in which respiratory insufficiency or failure can play an important role and is often the cause of death.

Influences on the Need for Respiratory Home Care

The American Medical Association's *Medical Management of the Home Care Patient: Guidelines for Physicians* states, "home care should be the 'first option'—preferred over hospitals, emergency departments, or nursing homes, whenever care needs can be safely met at home."[18] Several current trends and factors are present in the health care system that will influence the need for home respiratory services. These trends are all likely to increase the need for quality home services in the future.

Changes in Reimbursement. Changes in reimbursement will continue to affect the provision of respiratory home care. Managed care organizations, as well as Medicare and Medicaid, will continue to explore ways to stabilize or decrease costs while maintaining or improving outcomes. The home setting has proven to be a less costly setting for care than acute, subacute, and long-term care centers.[19-22] A wide variety of respiratory services can be safely delivered in the home. Many procedures currently delivered at higher levels of care are likely to move to the home. Pilot programs to provide home sleep studies,[23] pulmonary rehabilitation,[24-27] and exacerbation management[28-30] have demonstrated equivalent or better results at lower costs than hospital-based programs. Effective home care programs can even reduce the need for hospitalizations.[29-32]

Decreasing Availability of Home Health. With changes to the reimbursement structure of home health services, access to intermittent skilled visits by nurses, physical therapists, or occupational therapists has decreased. It has become increasingly difficult for patients to qualify for coverage of home health visits. A recent George Washington University study showed that 68% of hospital discharge planners report increased difficulty in initially obtaining home health services for Medicare beneficiaries.[33] As a result of reimbursement cuts from the Balanced Budget Act of 1997, nearly 2,500 Medicare-certified home health agencies have closed nationwide and 500,000 fewer beneficiaries were served in 1998 than 1997.[34] Many patients with home needs may not qualify under the current guidelines or the physician may forego home health services for fear of accusations of abuse or simple ignorance of the coverage guidelines.

Patient Preferences. Patients prefer to receive care in the comfort and safety of their own homes.[35] Provision of care in the home reduces exposure to infectious agents and allows for better rest. It has been demonstrated that quality of life is directly related to the patient's ability to remain at home and avoid the need for institutional care.[35]

Graying of the "Baby Boomers." In 1987, 12.2% of the population was age 65 years or older. By the year 2030 this percentage will increase to 25%. There are currently 35 million Americans age 65 years or older. Conditions more likely to occur in the elderly, such as COPD, are likely to increase as the population increases. With this, the need for quality respiratory home care will also increase.[36,37]

"Treatment to Prevention" Paradigm Shift. The training and skills of RTs have positioned them to take advantage of a paradigm shift in medicine, from *treatment* of disease to *prevention* of disease.[38] Spirometry has been demonstrated to predict debilitating lung disease decades in advance of the onset of symptoms, allowing an opportunity for aggressive intervention. An accelerated rate of decline not only predicts predisposition toward lung disease, but also heart disease,[39-42] stroke,[43] lung cancer,[44,45] cancers of all types, and premature mortality in general.[46] RTs are the personnel best trained to assist physicians in performing spirometry and other respiratory assessment.[47,48] They are also able to provide early intervention in the form of smoking cessation and other therapeutic options (eg, inhaled medications), and it is possible to deliver many of these interventions safely and cost-effectively in the home setting.[47,49-52]

Disease and Case Management. Though efforts at disease prevention will increase, chronic illness will continue

to be a primary concern within the health care system. Over half of the managed care dollars spent in the United States go toward treatment of the sickest 5% of patients and over 70% is directed to the sickest 10% of patients.[51,53] Programs that can demonstrate cost reductions while improving patient outcomes will be attractive to managed care providers, other insurers, employers, and patients, who all share in the cost of care. These programs have already demonstrated efficacy in disease states such as asthma and COPD.[54-58]

A major goal of such programs is to manage the patient in the least costly setting in which care can be safely and effectively administered, which is often the home.[19-22]

Earlier Dismissals from Acute Care. One of the emphases of managed care and Medicare's Diagnostically-Related Groups (DRG) system in controlling costs is the elimination of lengthy (and costly) hospitalizations. Under the previous "fee for service" arrangement, there was a financial incentive to keep patients in the hospital for as long as it took or until they were completely recovered. Now the goal is to manage the initial, most severe stage of the illness in the acute setting and to transfer the patient to a lower (and less costly) level of care as rapidly as possible without compromising outcomes or patient safety. This has translated into patients going home "quicker and sicker," which increases the need for quality home care to manage the more acutely ill home patient.[59] Cotton et al[30] found that a program of early discharge followed by home visits was as effective in preventing deaths and readmissions as is traditional in-patient management for uncomplicated exacerbations of COPD.

Advancing Technology. Advances in technology will continue to facilitate the move of care to the home. The cost of health care technologies continues to decrease, making them more accessible. Diagnostics and therapeutics once only available in the hospital setting are now routinely available in the home. Spirometers that fit in a pocket are available to provide accurate results, printouts, and even interpretation of results. Oximeters not much larger than the finger itself can provide instant readouts of oxygenation. The ongoing Sleep Heart Health Study has demonstrated that home polysomnography is a viable alternative to hospital-based studies.[60]

Telemedicine is certain to affect the home setting as well. It is not unreasonable to picture a scenario where a homebound patient is assessed and managed by the physician via monitoring transmitted over telephone lines or other modes of electronic transfer. Transmission of video signals, electrocardiography data, lung sounds, spirometry data, sleep data, and other physiologic data is no longer hypothetical; it is available today. Advancing technology

is certain to improve the quality of these data while decreasing costs.

These and other factors will continue to make the home setting more important in the overall care of patients requiring respiratory services. Regrettably, in the face of the increasing need for quality respiratory home care, there are substantial changes in reimbursement that threaten to limit the availability of professionals to provide this care.

How Is Optimal Respiratory Home Care Delivered?

Team Approach

As in other specialties, such as pulmonary rehabilitation, optimal care is achieved by a team effort. This team includes the patient, the patient's family and/or friends who are involved in care, the physician, the physician's staff, and ancillary health care personnel who provide in-home services (Table 2). Other community services (eg, "Meals On Wheels" and home aides) may also be required to make the home environment more conducive to patient care. For optimal care to occur, it is essential that adequate support (eg, caregivers) and resources (eg, medical equipment) are available to the patient. Failure to provide this support is likely to result in the need for placing the patient in higher, more expensive levels of care (eg, nursing facilities) or to increase the frequency of hospitalization.[59,61,62]

Respiratory Therapists in the Home

RTs are the ancillary health care personnel most likely to be available for many patients with chronic lung dis-

Table 2. Components of the "Respiratory Home Care Team"

Patient and caregivers
  Patient
  Spouse
  Other family members
  Friends/neighbors

Physician's office
  Physician
  Nursing staff
  Other clinical staff
  Non-clinical staff

Ancillary health care professionals and paraprofessionals
  Respiratory therapist
  Home health nurse
  Occupational therapist
  Physical therapist
  Registered dietitian
  Dismissal coordinators
  Home aides

ease. As stated previously, home health visits by nurses and physical therapists are being curtailed by changes in the reimbursement structure.[33,34] Because many of these patients require durable medical equipment (eg, nebulizers, oxygen, ventilators), RTs employed by home medical equipment and respiratory therapy (HME/RT) providers perform home visits to these patients. RTs are employed by HME/RT companies because RT expertise is required for setting up and maintaining home respiratory equipment. On less complex pieces of equipment (eg, nebulizer), the RT visit may be a one-time event at the set-up, whereas for more complex equipment (eg, oxygen, ventilators) home visits may be made on a regular and ongoing basis.

Because the RT is already visiting the patient's home, there is an opportunity for the RT to function as a support to the physician in optimizing the home respiratory care provided. The training, skills, and experience of the RT can be invaluable to both the patient and the physician in a number of ways.

Patient Education. Education of the patient, the family, and other caregivers is an essential element of effective disease management. RTs are well-equipped to provide training on lung function, pathophysiology, cardiopulmonary medications, breathing and cough retraining, use and care of equipment, smoking cessation, recognition of signs of an exacerbation, the importance of regular activity or exercise, and other topics pertinent to patient care.[49,63] Patients who receive education are better equipped to participate in their own care (ie, collaborative self-management[64]) and more likely to remain compliant with the treatment ordered by the physician.[65-68] Demands on the time of the physician make it difficult, if not impossible, for the physician to spend the time required for adequate education in these subjects.

Monitoring Response to Therapy. By observing and assessing the patient on an initial visit or during ongoing visits, the therapist is able to evaluate the patient's response to therapy. For example, if the patient continues to exhibit symptoms of bronchoconstriction (eg, wheezing, coughing, dyspnea) after the implementation of bronchodilator therapy, this should be reported to the physician so that alterations can be made to the bronchodilator therapy, such as adding additional medications,[69] increasing dose or frequency,[70] or changing delivery methods.[71] Failure to do so could result in suboptimal management of air flow obstruction, decreased function, the need for additional hospitalization, and increased total cost of care.[69-71]

Recognizing and Responding to Complications and Adverse Reactions. With most home therapies there is the potential for complications or adverse reactions. The patient or the patient's family may disregard the importance of seemingly benign reactions to therapy as "not important enough" to call the doctor. Minor complications such as a dry or sore nose may lead to noncompliance with oxygen therapy. More serious adverse reactions such as pneumothorax due to positive pressure therapy (eg, ventilation) may even place the patient's well being at risk. A skilled therapist will not only recognize the importance of such problems but can offer solutions to improve compliance and reduce risk to the patient.

Equipment Monitoring. It is important that respiratory equipment be properly monitored for safe and effective operation. Patients typically do not have the ability to do so on their own because of a lack of knowledge and specialized equipment necessary to monitor the operation of the equipment (eg, oxygen analyzers, pressure manometers). It is our experience that when oxygen concentrators go unmonitored they may continue to "run" and give the perception of working correctly, but on examination with an oxygen analyzer it is revealed that they are dispensing nothing more than room air. This has been demonstrated in countries where oxygen concentration is monitored less frequently.[72]

Education and Monitoring of Infection Control Procedures. Patients should be educated in proper cleaning and infection control procedures and then monitored for compliance. The consequences of inadequate infection control can be serious. Without proper maintenance, a device meant to help a patient could actually become the instrument that causes repeated infections, exacerbations, and even hospitalizations.[73]

Adjusting Therapy Based on Response. Just as in the acute care setting, it is often desirable to allow the therapist to adjust therapy within physician-defined guidelines based on patient response. This places the therapist in the role of the "physician extender" in much the same manner that therapist-driven protocols do in the acute care setting. It allows for more appropriate therapy while minimizing inconvenience to the physician. Having the physician perform these procedures would be burdensome and logistically difficult. A common example of physicians giving home RTs this level of responsibility is in adjusting oxygen therapy (eg, titrating flow to maintain pulse-oximetry-measured blood oxygen saturation [S_{pO_2}] over 90%) and especially in noninvasive positive pressure ventilation, where titration of multiple ventilation variables (eg, inspiratory positive airway pressure, end-expiratory positive airway pressure, fraction of inspired oxygen, backup rate, rise time, and inspiration-expiration ratio) is commonly performed in the home by the therapist within established guidelines.

Exacerbation Prevention. By educating patients and their caregivers on the early signs of exacerbation and by monitoring patients on regular visits for signs and symptoms of deterioration, the RT can help prevent exacerbations or at least quickly intervene so as to avoid hospitalization. In COPD, exacerbations are generally linked to infections or heart failure, both of which can be managed more effectively when recognized and treated in their earliest stages. Early intervention can spell the difference between managing the patient at home (eg. adding antibiotics, a steroid burst, and more aggressive bronchial hygiene) or providing the same care in a more costly acute care setting.[74]

Environmental Assessment. The home setting can have a dramatic impact on the condition of the respiratory patient. The effects of a poor home environment may include exposure to common allergens (eg. dust mites, cockroach excrement, pet dander, mold), inhaled irritants (eg. tobacco smoke, cooking fumes, perfumes, cleaners), lack of adequate resources (eg. financial, nutritional), improper support systems (eg. caregivers), inadequate mechanical systems (eg. electrical, clean water, air conditioning), difficult physical circumstances (eg. multiple stairs, unstable flooring), and even abusive situations (eg. neglect, physical abuse).

The American Medical Association recognizes the importance of in-home assessment, stating that

> For most patients, in-home assessments are preferable and may be crucial to fully understand a patient's care needs. In-home assessments can be highly efficient ways to save time in diagnosis, medical decision-making, and communication among all team members. These assessments may be performed by physicians or by other health care professionals who are in close communication with the physician, depending upon the circumstances.[18]

Patient Compliance. Noncompliance with therapy is a common problem with respiratory patients. Estimated noncompliance with medications in the elderly has been estimated at 40–75%.[75] Pediatric populations show similar results.[76] Investigations of compliance with long-term oxygen therapy (LTOT) suggest that compliance rates are comparable. Six studies have found compliance rates of 45–74% (45%, 55%, 56%, 65%, 65%, 74%).[70,77-81] Addressing noncompliance with oxygen therapy has been cited as a primary recommendation in two recent consensus conferences reviewing the current status of long-term oxygen therapy.[82,83]

In chronic disease, noncompliance with therapy has been shown to place the patient at higher risk of poor illness management, increased symptoms, poorer functional status, more missed days from work and school, more frequent exacerbations, and even higher mortality.[84,85] With LTOT, failure to use oxygen as ordered can affect both morbidity and mortality, as oxygen is the only form of therapy shown to extend life span, and failure to use oxygen for an adequate number of hours per day has been shown to affect survival.[86,87] Physicians are frequently criticized for the lack of time spent in educating patients on why they are using a therapy and how it should be used.[88] Monitoring the patient for adherence to the physician's orders while providing education and support can both prevent noncompliance and help reestablish compliance where problems exist.[69,80]

Several factors have the potential to affect patient compliance with therapy. Pepin et al suggested that factors associated with effective use of oxygen are (1) initial prescription for 15 hours or more per day, (2) supplementary education on oxygen, (3) cessation of smoking, (4) use of oxygen in all domestic situations (toilet, meals, leisure, etc), and (5) absence of adverse effects from oxygen treatment. Their conclusion was that attention to such factors could optimize oxygen prescription and constitute goals for patient education.[77]

A recent report from the Office of the Inspector General found that, although almost all patients with home oxygen stationary systems used them, 13% reported never using their portable systems.[89] The two most probable reasons for this noncompliance are (1) the patients do not need the portable systems or (2) the patients have continuing need and are simply not using the systems as appropriate to their needs. Since Medicare requires documentation of medical need and the need for supplemental oxygen generally increases during activity, the latter explanation is more likely. In either case, additional monitoring and education by RTs has the potential to reduce this waste of resources.

Improved Allocation of Resources. Although data on the misallocation of respiratory home care resources in the United States are sparse,[89] data from other levels of care and other countries suggest that the potential for misallocation is high. Misallocation of resources can take multiple forms in the home, including:

1. patients receiving respiratory services who do not have documented medical need

2. patients receiving respiratory services who had documented medical need during an acute episode but no longer require them after resolution of an acute process

3. patients who have medical need but are not receiving services

4. patients receiving services prescribed at suboptimal settings, dosages, delivery methods, or frequency

5. patients receiving services for which there is documented medical need but who are noncompliant in using the services

All forms of misallocation have the potential to increase the cost of care. In the United States, because there is a requirement for documentation of medical need for most respiratory home care services (eg, oxygen requirements), it is unlikely that a large number of patients fall into the first category.

Experts at multiple consensus conferences have suggested that patients may fall into the second category more frequently.[82,83,90-93] Consequent to increasing pressure for early hospital dismissal, patients are being dismissed earlier, while still recovering from exacerbations of chronic states or acute illness. The potential exists for physicians to prescribe respiratory services (eg, oxygen) based on testing done during an acute phase, only to have the patient continue on the service long after the acute phase, when medical need no longer exists.

In Spain, Farrero et al found that monitoring with oximetry during home visits led to the withdrawal of LTOT from 20 of 128 patients.[94] This underscores a point made by the Fifth Oxygen Consensus Conference:

> Patients who are discharged from hospitals following an exacerbation with unstable respiratory disease requiring oxygen therapy should be recertified after the initial 90 days of therapy with long-term oxygen by repeat arterial blood gas analysis or oxygen saturation measurements. These measurements are medically necessary for the physician to evaluate the course of the disease and to make adjustments to oxygen flow or to discontinue oxygen if it is no longer necessary. Once the need for LTOT is established, repeat measurements of arterial blood gases or saturation are not necessary or justifiable.[83]

The American Medical Association suggests that a number of patients may fall into the third category: those who have medical need for services but do not currently receive them. The American Medical Association states:

> An estimated 20% of patients over 65 have functional impairments with related home care needs. Their physicians may be unaware of these needs during the typical office visit. For some relatively functional patients, home care needs may be adequately defined in an office setting. However, for most patients, in-home assessments are preferable and may even be critical. In-home assessments can be highly efficient ways to save time in diagnosis, medical decision-making, and communication among all team members. These assessments may be performed by physicians or by other health care professionals who are in close communication with the physician, depending upon the circumstances.[38]

Home RTs are in an excellent position to perform home assessments and communicate the results to the physician.

It is estimated that approximately half of COPD patients have been identified. The National Lung Health Education Program, a program endorsed by multiple physician, clinical, and governmental groups, is focused on improving identification of COPD through education of primary care physicians on spirometry testing. Certainly, increased home assessment by RTs can result in better identification, which is likely to increase the identified need for respiratory home care. It is important to note, however, that this increased identification of need does not translate directly to increased total costs. If better home management is available, the need for higher and costlier levels of care may be prevented.[20,21,31,32]

Studies suggest that many patients may fall into the fourth category: those prescribed treatment at suboptimal settings, dosages, delivery methods, or frequency. A ready example is LTOT. Consensus conference participants cited the need for individualized adjustment of the oxygen prescription, something that rarely happens in practice.[82,83] Evidence suggests that a substantial percentage of patients on home oxygen may not be adequately corrected by their current oxygen flows. Morrison et al found that in patients with daytime arterial partial pressure of oxygen ≥ 60 mm Hg, S_{pO_2} was more than 90% for an average of 78% of the time. In patients with daytime arterial partial pressure of oxygen of < 60 mm Hg, S_{pO_2} was more than 90% for an average of 69% of the 24-hour period.[95]

Sliwinski et al found that despite having an average S_{pO_2} of 94% at the beginning of the recording, patients on oxygen spent an average of 6.9 hours below S_{pO_2} of 90%, with a minimum S_{pO_2} of 61%. Most desaturations came during sleep and naps. The study concludes, "The oxygen flow prescribed, based on blood gas measurements at rest, did not protect 85% of the patients studied from deep falls in S_{pO_2} during daily life."[96]

Carroll et al reported that 4 of 10 patients showed substantial desaturation, with S_{pO_2} decreasing to below 90% for periods of 15–47% of the monitoring time.[97] Gorzelak found that nocturnal oxygen desaturation affects prognosis in COPD patients, despite long-term oxygen treatment.[98]

Therapist-driven protocols for monitoring and titrating oxygen flows to an individualized prescription based on resting, activity, and nocturnal needs would probably correct this common error in prescription. To obtain maximum benefit from oxygen therapy, it is important to correct oxygenation at all times. In the Nocturnal Oxygen Therapy Trial, hypoxemic patients who used oxygen an average of 19 hours per day (and presumably whose oxygen levels were corrected for longer periods) lived significantly longer than those averaging only 12 hours of daily use.[99]

The fifth and final category—those with medical need but noncompliant with care—constitute a substantial problem. Every attempt should be made to improve compliance

through education and patient support. In the event that the patient chooses to remain noncompliant despite these efforts, the physician should decide whether it is prudent to leave equipment in place based on the current patient use patterns.

In other settings, data indicate that RTs can decrease misallocation of services, especially when therapist-driven protocols are in place.[100–111] Multiple studies show that the use of therapist-driven protocols results in improved outcomes and decreased cost of care. This allows the physician more time to perform other duties and replaces house staff (eg, interns, residents), who are less available because of current trends in physician education and training. It is reasonable to expect that the use of RT-driven protocols would result in similar benefits in the home setting as well.

Monitoring Psychosocial Status. Clinically important depression and other psycho-emotional disorders are common among patients with chronic illness, including COPD.[112–114] Though RTs are not qualified to directly address those issues, they can certainly recognize their impact on care and bring them to the physician's attention. The therapist may indirectly assist in preventing or managing these problems by addressing commonly linked issues such as inactivity, frequent exacerbations, fears from ignorance of disease process, and lack of social interaction, as has been demonstrated in pulmonary rehabilitation programs.[115–117]

The patient's condition itself can have substantial impact on the patient's cognitive status. Hypoxemia, hypercapnia, and poor sleep quality are common in chronic pulmonary disease and directly affect the patient's concentration, memory, reasoning, and alertness. By monitoring for the signs and symptoms of these conditions and reporting them to the physician along with appropriate recommendations for intervention (eg, inhaled bronchodilators, oxygen therapy, noninvasive ventilation), these problems can be effectively managed.

"Physician Extender." By working under the direction of the physician in monitoring the patient for response to therapy, reporting important findings to the physician, and making appropriate recommendations for adjusting the current plan of care, the RT assumes the role of a "physician extender" in the home setting. Although it is desirable for the physician to directly visit the patient's home, it is logistically difficult (if not impossible) for today's physician to visit every patient's home, even on a one-time basis. Home care can be very effective in identifying new problems that may not be discovered in the physician's office.[18,118–120]

The American Medical Association recommends[121] that each home care visit should include:

- a brief assessment of the overall effectiveness of the comprehensive home care program
- assessment of patient and caregiver interactions and satisfaction with the home care program
- identification of any new problems
- notification of appropriate team members for follow-up of new problems
- encouragement and reinforcement of instructions from other team members

The home RT is in an ideal position to provide this type of feedback to the physician and other team members (eg, ancillary health personnel [see Table 2] who provide in-home services). The therapist can also follow up on problems or concerns noted by "nonclinical" personnel and relay pertinent information to the physician. For example, during the delivery of a hospital bed, the delivery technician may note on the plan of service that the patient reported shortness of breath with minimal activity. As the physician may be unaware of this problem, the RT should gather appropriate information and relay it to the physician, resulting in appropriate intervention. To ignore such a finding would be irresponsible.

An essential element of this physician extender relationship is trust. Trust is built between the physician and therapist based on the demonstrated knowledge and skills of the therapist. If the therapist faithfully provides information to the physician that is accurate, reliable, concise, and important to the overall care of the patient, trust will grow. The home RT must have strong assessment skills and the ability to communicate effectively with the physician and patient. A trusting relationship based on multiple positive contacts is conducive to the physician allowing the therapist a growing role in the management of home care patients.

Physicians must likewise give therapists an opportunity to prove their value in the home setting. This is done by allowing therapists to actively participate in the home management of the patient and by being open to input and ideas submitted by the therapist. The physician must realize that by working effectively with the therapist, care can be improved to everyone's benefit.

Benefits of Respiratory Home Care

The benefits of respiratory home care have been well established by a number of authors, all aimed at improving outcomes through comprehensive home respiratory services. The addition of evaluation and follow-up visits by RTs in the South Hills Health System project decreased the need for hospitalizations in COPD patients from 1.28 admissions per patient the year prior to the program to 0.48 the year following. Average length of hospital stay also decreased, from 18.25 days to 6.09 days.[22]

The "Respi-Care" program targeted chronic respiratory patients with histories of frequent hospitalization. By providing comprehensive home care, including RTs, to 17 patients with end-stage respiratory disease, they were able to decrease hospitalizations from 88 to 53 in equal time periods before and after the institution of the program. Hospital days decreased from 1,181 before the program to 667 during the program, and emergency department visits decreased from 105 to 64. Total costs savings were $328 per patient per month (1991 data).[20]

Zajac demonstrated significant improvement in outcomes by using RTs in a "Respiratory Wellness Program" for managed-care organization members with asthma and COPD. A cohort of 370 COPD patients showed a 70% reduction in hospital days and emergency department visits. More than 95% of patients expressed satisfaction with the program.[123]

Warburton et al demonstrated improvements in managed care members with asthma. Participants showed improvements in peak flow meter use, personal best peak flow, lost productive days, lost productive days for child care, use of inhaled corticosteroids, shortness of breath, chest tightness, night awakenings, member-reported hospital admission rates, member-reported average length of stay, annual admission rates, semi-annual admission rates, and average length of stay.[123]

Weber et al demonstrated a reduction in health care utilization in 451 asthma patients who completed a "respiratory wellness program" that included RT home visits for patient instruction, self-care instruction, and patient assessment. There was also a trend toward a reduction in disease severity for patients who had participated for 300 days or more.[56]

In a separate project, Weber et al used RTs in an in-home program to reduce health care utilization, and improve quality of life and functional status in 349 COPD patients. On average, health care utilization (sum of hospital days and emergency department visits) dropped from 3.4 to 0.4 in Stage 1 COPD, from 3.6 to 1.1 in Stage 2 COPD, and from 6.1 to 1.4 in Stage 3 COPD.[124]

Hendon and Kageler used an RT-delivered disease management program to reduce health care utilization in asthma and COPD patients. Fifty-seven patients with asthma or COPD showed significant reductions in emergency room visits (1.75 to 0.32), hospitalizations (0.96 to 0.11), hospital days (5.12 to 0.44), and lost productive days (6.55 to 1.58), with improvement in quality of life.[125]

Roglieri et al demonstrated that RTs can be effective case managers with asthma patients. Their program used RTs to provide home visits for patient assessment, environmental assessment, education, self-care instruction, and action plan implementation. Patients who completed the program were more likely to adhere to asthma guidelines, including increased use of anti-inflammatory drugs.[126,127]

Through the use of chronic disease management, Tiep et al demonstrated a reduction in hospitalizations (treatment group: 22 pre-referral vs 17 post-referral, control group 34 pre-referral vs 46 post-referral, p < 0.05) and total hospital days (treatment group: 177 pre-referral vs 91 post-referral; control group: 221 pre-referral vs 251 post-referral, p < 0.05). They estimated savings in hospital charges of $360,000 for the treatment group (n = 55) and $700,000 for the group as a whole (n = 109). The yearly cost of the program was $300 per participant per year.[?]

Fields et al demonstrated substantial cost reductions in technology-dependent children with the use of a comprehensive respiratory home care program. For 6 ventilator-dependent children, the savings were $79,074 ± $26,558 per patient per year. For 4 oxygen-dependent children, the savings were $83,187 ± $25,028 per patient per year.[19]

Bach et al had similar results in 20 adult ventilator-assisted individuals. The daily cost of caring for these patients in the home was $235.13 ± $56.73, whereas Medicaid reimbursement for respiratory rehabilitation units was $648–$719 per day.[24]

Others have found similar reductions in the need for hospitalizations, outpatient care, and total costs with comprehensive home care programs.[31,32,128,129]

How Does Reimbursement Affect the Quality of Care?

Influence of Equipment Reimbursement Methods on Quality of Care

Congress recognized the importance of close follow-up when they crafted the definition of "Frequent and Substantial Servicing" in establishing payment categories under the 6-point plan currently used in reimbursement of home medical equipment under Medicare Part B:

> This class of items [requiring frequent and substantial servicing] would include those that are technologically sophisticated and require frequent monitoring or adjustment in order to make sure they are functioning properly or being properly utilized by the patient.[130]

Common sense dictates that you cannot separate home and patient variables from the equipment when ensuring that the equipment is properly utilized by the patient. All of the factors discussed above have direct impact on whether proper utilization will occur. Appropriate follow-up by qualified personnel is essential to ensuring that patients comply with physician orders. With oxygen therapy, non-invasive ventilation, invasive ventilation, and other technologically sophisticated home therapies, it is essential that proper follow-up be available. Failure to provide such

care ensures generally poor compliance and suboptimal patient outcomes.

A quick review of equipment that has been moved from the "frequent and substantial servicing" category to the "capped rental" category of Medicare's reimbursement system can teach an important lesson. Under capped-rental, items are rented for a predetermined period of time (13 or 15 months, depending on the patient's choice of purchase/rent option), at which time payment is "capped." Because of the limited reimbursement for these items, ongoing follow-up by professionals is typically unavailable. Continuous positive airway pressure devices were moved into this category in 1995. Patients have demonstrated poor compliance (less than 50%) with continuous positive airway pressure, as monitored by the devices' hour meters.[131] Interventions such as those discussed above have been demonstrated to improve compliance when available.[67] Certainly the case can be made that moving these devices (eg, continuous positive airway pressure, bilevel positive airway pressure, nebulizers) to capped rental was a mistake that should be reversed.

Effect of Recent Cuts

The implementation of stringent guidelines on qualification for home health visits has dramatically decreased the availability of nurses and other health professionals for home visits via the home health agency.[33] Furthermore, implementation of a prospective payment system has driven many home health agencies out of business.[34]

The Health Care Financing Administration has enacted several cuts in reimbursement for home respiratory equipment, including oxygen, nebulizer medications, and other services, citing perceived overpayment for those services based on the rates being paid by other providers (eg, Veterans Affairs Department). The validity of their comparisons has been called into question by the Fifth Oxygen Consensus Conference.[83]

These substantial decreases in Medicare payments for home oxygen and other home medical equipment services have caused many home medical equipment providers to reevaluate the feasibility of employing RTs. Because there is typically no direct payment for the services of the home RT, the cost of the clinicians is absorbed by the HME/RT providers. As cuts continue and profit margins decrease, it becomes increasingly difficult to justify these added costs. In response to these cuts in reimbursement, many HME/RT companies have decreased the frequency of follow-up visits by RTs to home oxygen patients. Some have even eliminated these visits. Many patients who once enjoyed monthly visits by RTs are now only visited quarterly (or less often), and at times by nonclinicians. Further cuts in equipment reimbursement via competitive bidding, the application of inherent reasonableness, and other reductions

in equipment reimbursement are currently being considered and proposed. It is also being proposed to move more of these devices (eg, noninvasive ventilation, oxygen therapy) out of the frequent and substantial servicing category and into capped-rental. The impact on patient care, patient safety, and patient outcomes would be devastating.

Unless these trends in reimbursement cuts are reversed, home care is likely to degrade into a low service, "fast food" commodity. Service will be cut to a level that negatively impacts patient care. Numerous examples of poor patient care secondary to service cutbacks have been cited by patients and clinicians during recent conferences.[83] Home medical equipment and services will be provided by individuals with inadequate training to provide the level of support required by patients. This is at a time when all trends point to a demand for higher quality home medical care.

Discussion

Respiratory home care faces a tremendous paradox: during a time of increasing need there is decreasing reimbursement for services. Providers must respond to a number of challenges, including:

• Providing more definitive data on the benefits of respiratory home care to patient outcomes (eg, function, quality of life, decreased hospitalizations, decreased total costs). Substantive data on the benefits of respiratory home care, especially in reducing costs, would be a major step toward securing reimbursement for the services themselves rather than just the equipment.

• Improved allocation of respiratory services (both human and equipment) to improve outcomes while maintaining or reducing total cost. Providers must be judicious and efficient in their use of skilled personnel (eg, RTs).

• Increasing credibility. Providers have faced criticism concerning overpayment, overutilization, and other fraud and abuse. Clear clinical guidelines must be developed for the proper application of respiratory home care services. Therapist-driven protocols, which have reduced misallocation in the acute care setting, may provide an effective mechanism in the home as well.

Summary

There are many opportunities for the physician and home RT to work together to provide high quality, home respiratory care. The skills of the RT are well suited to:

· reduce misallocation of respiratory services
· assess the patient's respiratory status
· identify new or as yet unidentified problems and needs of the patient
· evaluate the effect of the home setting
· educate the patient on proper use of the equipment

- monitor the patient's response to therapy, including complications of therapy
- monitor the equipment for proper function
- monitor for appropriate infection control procedures
- make appropriate recommendations for changes to current therapy
- adjust therapy under the direction of the physician

By working together, everyone benefits. The physician benefits from increased access to important information concerning the patient and the impact of the home environment. This is accomplished in a much more efficient and cost-effective manner than the physician visiting the home, and allows the physician to focus on higher-level tasks that require direct physician oversight. The RT benefits by being able to function in a way more valuable to the physician and the patient. By using the skills they have worked hard to achieve, RTs have greater job satisfaction than when they are placed in roles of delivering rote and repetitive care that does little to challenge their skills and fails to have an important impact on care.

Finally, and most importantly, the patient benefits by receiving closer monitoring, better education, and the support needed to allow for safe and effective home respiratory care. With the patient receiving more appropriate care, quality of life improves, the need for hospitalization decreases, and costs and inconvenience associated with the disease are lessened. Thus, the previously stated goals of home respiratory care—to improve the patient's physical well being, potential for productivity, and self-sufficiency within the individual's limitations—are achieved.

REFERENCES

1. American Association for Respiratory Care. Position Statement: Definition of respiratory home care, 1991. Available at http://www.aarc.org/resources/position_statements/drhc.html (Accessed March 23, 2001)
2. American Society of Anesthesiologists. Statement of support for respiratory care practitioners, 1996. Available at http://www.aarc.org/advocacy/resources/physician_letters/asa_letter.jpg. (Accessed Feb 15, 2001)
3. National Association for Medical Direction of Respiratory Care. NAMDPC Statement: Non physician providers of respiratory care services, 1998. Available at http://www.aarc.org/advocacy/resources/physician_letters/namdrc.gif. (Accessed Feb 15, 2001)
4. American College of Chest Physicians Section on Respiratory Care. Resolution: Role of respiratory care practitioners in the delivery of respiratory care services, 1997. Available at http://www.aarc.org/advocacy/resources/physician_letters/accp_letter.gif. (Accessed Feb 15, 2001)
5. California Thoracic Society. Position Paper: Respiratory care at alternate (out of hospital) sites, 1997. Available at http://www.aarc.org/advocacy/resources/physician_letters/cts_paper.html. (Accessed Feb 15, 2001)
6. American Medical Association white paper on elderly health. Report of the Council on Scientific Affairs. Arch Intern Med 1990;150(12):2459-2472.
7. American Thoracic Society. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1995;152(5 Pt 2):S77-S121
8. National Heart, Lung, and Blood Institute. Morbidity and mortality chartbook on cardiovascular, heart, and lung diseases, 2000. Available at http://rover.nhlbi.nih.gov/resources/docs/cht-book.htm (Accessed Mar 7, 2001)
9. Hurd SS. International efforts directed at attacking the problem of COPD. Chest 2000;117(5 Suppl 2):336S-338S
10. National Center for Health Statistics (1995). Current estimates from the National Health Interview Survey, 1994. Vital Health Stat 10(193). Hyattsville MD, author. Table 62.
11. Surveillance for asthma—United States, 1960-1995. MMWR Surveillance Summaries, April 24, 1998;47(SS-1):1-28. Available at http://www.cdc.gov/epo/mmwr/preview/mmwrhtml/00052262.htm. (Accessed Mar 7, 2001)
12. Monthly vital statistics report, August 14 1997;46(1), table 6. National Center for Health Statistics. Available at http://www.cdc.gov/nchs/data/mvsr/mvsr46_1.pdf. (Accessed Mar 7, 2001)
13. HHS targets efforts on asthma. Fact sheet. Dept of Health & Human Services, Bethesda, Maryland, May 21, 1998. Available at http://www.hhs.gov/news/press/2001pres/01fsasthma.html. (Accessed Mar 7, 2001)
14. Asthma: a concern for minority populations. National Institute of Allergy and Infectious Diseases, January, 1997. Available at http://www.8004asthma.com/asthma-minority%20concern.htm. (Accessed Mar 7, 2001)
15. Monthly vital statistics report. Annual Summary of Births, Marriages, Divorces, and deaths: United States, 1990. US Center for Disease Control/National Center for Health Statistics. Department of Health and Human Services. Aug 28 1991; 39(13):1-28.
16. National Commission on Sleep Disorders Research. William C Dement. Chair. Wake Up America. A National Sleep Alert. Volume One: Executive Summary and Executive Report. US Government Printing Office. Doc No 1993-342-299/30631, January, 1993
17. A question of care. Modern Healthcare 1996;26:122-129
18. American Medical Association. Medical management of the home care patient, guidelines for physicians, 2nd ed. 1998.
19. Fields AI, Rosenblatt A, Pollack MM, Kaufman J. Home care cost-effectiveness for respiratory technology-dependent children. Am J Dis Child 1991;145(7):729-733.
20. Haggerty MC, Stockdale-Woolley R, Nair S. Respi-Care: an innovative home care program for the patient with chronic obstructive pulmonary disease. Chest 1991;100(3):607-612.
21. Bach JR, Intintola P, Alba AS, Holland IE. The ventilator-assisted individual: cost analysis of institutionalization vs rehabilitation and in-home management. Chest 1992;101(1):26-30.
22. Roselle S, D'Amico FJ. The effect of home respiratory therapy on hospital readmission rates of patients with chronic obstructive pulmonary disease. Respir Care 1982;27(10):1194-1199.
23. Sergi M, Rizzi M, Greco M, Andreoli A, Bamberga M, Castronovo C, Ferini-Strambi L. Validity of diurnal sleep recording performed by an ambulatory device in the diagnosis of obstructive sleep apnoea. Respir Med 1998; 92(2):216-220.
24. Bauldoff GS, Hoffman LA, Sciurba F, Zullo TG. Home-based, upper-arm exercise training for patients with chronic obstructive pulmonary disease. Heart Lung 1996 25(4):288-294
25. Strijbos JH, Postma DS, van Altena R, Gimeno F, Koeter GH. A comparison between an outpatient hospital-based pulmonary rehabilitation program and a home-care pulmonary rehabilitation program in patients with COPD. a follow up of 18 months. Chest 1996; 109(2):366-372.
26. Strijbos JH, Postma DS, van Altena R, Gimeno F, Koeter GH. Feasibility and effects of a home-care rehabilitation program in patients

with chronic obstructive pulmonary disease. J Cardiopulm Rehabil 1996;16(6):386–393.

27. Maltais F, Debigare R, Bourbeau J, Rouleau M, Lacasse Y, Beaupre A, et al. Characteristics, acceptability, and feasibility of a home-based exercise training program in COPD (abstract). Am J Respir Crit Care Med 1999;159(3 Pt 2):A311.

28. Gravil JH, Al-Rawas OA, Cotton MM, Flanigan U, Irwin A, Stevenson RD. Home treatment of exacerbations of chronic obstructive pulmonary disease by an acute respiratory assessment service. Lancet 1998;351(9119):1853–1855.

29. Hernandez C, Celorrio N, Gavela E, Collyment B, Farrero E, Fernandez I, et al. Efficacy of home hospitalization in COPD patients admitted in the emergency room for an acute exacerbation (abstract). Am J Respir Crit Care Med 2000;161(3):A812.

30. Cotton MM, Johnson MK, Stuart C, Stevenson RD. A randomized controlled trial of early discharge with domiciliary support versus conventional in-patient management for exacerbations of chronic obstructive pulmonary disease requiring hospital admission (abstract). Am J Respir Crit Care Med 1999;159(3 Pt 2):818.

31. Clini E, Vitacca M, Foglio K, Simoni P, Ambrosino N. Long-term home care programmes may reduce hospital admissions in COPD with chronic hypercapnia. Eur Respir J 1996;9(8):1605–1610.

32. Miles-Tapping C. Home care for chronic obstructive pulmonary disease: impact of the Iqalut program. Arctic Med Res 1994; 53(4): 163–175.

33. National Association for Home Care. News release. Report confirms Medicare cuts hurting frailest home care patients. Jan 18, 2000. Available at http://www.nahc.org/NAHC/NewsInfo/00nr/medcutrpt.html. (Accessed Mar 7, 2001.)

34. National Association for Home Care. News release: HCFA data reflect staggering drop in home health outlays. Oct 20, 1999. Available at http://www.nahc.org/NAHC/NewsInfo/99nr/hhout.html (Accessed Mar 7, 2001).

35. Effect of exacerbation on quality of life in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1998 May;157(5 Pt 1):1418–1422.

36. US General Accounting Office. Long-term care: projected needs for the aging baby boom generation. GAO/HRD,91:86 Washington DC: US Accounting Office, Jun 1991.

37. US Department of Commerce. Bureau of the Census. Statistical abstract of the United States. 11th edition, 1991.

38. The Lewin Group. Respiratory care practitioners in an evolving health care environment. AARC Times 1997;21(Dec):8–19.

39. Petty TL. Office spirometry and the cardiac patient. Practical Cardiology Sept 1978:41–51.

40. Kannel WB, Seidman JM, Fercho W, Castelli WP. Vital capacity and congestive heart failure: the Framingham study. Circulation 1974; 49(6):1160–1166.

41. Rodriguez BL, Masaki K, Burchfiel C, Curb JD, Fong KO, Chyou PH, Marcus FB. Pulmonary function decline and 17-year total mortality: the Honolulu Heart Program. Am J Epidemiol 1994;140(5): 398–408.

42. Tockman MS, Pearson JD, Fleg JL, Metter EJ, Kao SY, Rampal KG, et al. Rapid decline in FEV₁ a new risk factor for coronary heart disease mortality. Am J Respir Crit Care Med 1995;151(2 Pt 1): 390–398.

43. Strachan DP. Ventilatory function as a predictor of fatal stroke. BMJ 1991;302(6768):84–87.

44. Skillrud DM, Offord KP, Miller RD. Higher risk of lung cancer in chronic obstructive pulmonary disease: a prospective, matched, controlled study. Ann Intern Med 1986;105(4):503–507.

45. Tockman MS, Anthonisen NR, Wright EC, Donithan MG. Airways obstruction and the risk for lung cancer. Ann Intern Med 1987; 106(4):512–518.

46. Petty TL. John Hutchinson's mysterious machine. Transactions of the American Clinical and Climatological Association 1986, 98.11–20.

47. Czachowski R. Study finds respiratory care instruction very limited in nursing schools. AARC Times 1994;18(Aug):99–108.

48. AARC Clinical Practice Guideline: Spirometry, 1996 update. Respir Care 1996, 41(7):629–636.

49. AARC Clinical Practice Guideline. Providing patient and caregiver training. Respir Care 1996;41(7):658–663.

50. AARC Clinical Practice Guideline. Selection of a device for delivery of aerosol to the lung parenchyma. Respir Care 1996;41(7):647–653.

51. AARC Clinical Practice Guideline. Selection of aerosol delivery device. Respir Care 1992;37(8):891–897.

52. Moran D. Lecture at the National Association for Medical Direction of Respiratory Care Annual Meeting. LaJolla CA, Mar 1999.

53. Sullivan SD, Ramsey SD, Lee TA. The economic burden of COPD. Chest 2000;117(2 Suppl):5S–9S.

54. Myers TR, Chatburn RL, Kercsmar CM. A pediatric asthma unit staffed by respiratory therapists demonstrates positive clinical and financial outcomes. Respir Care 1997; 43(1):22–29.

55. Volsko T. A Pediatric asthma clinic pilot program reduces emergency department visits, hospitalizations, and cost of care. Respir Care 1998;43(2):107–113.

56. Weber K, Hendon B, Kagerler V. Changes in disease severity and health care utilization following a respiratory wellness program for asthma (abstract). Chest 1998,114:299S.

57. Liep BL, Marnett M, Robles M, Sadana G, Lewis D, Nicholson D, et al. Chronic disease management reduces hospitalizations in chronic lung disease (abstract). Chest 1998,114:255S.

58. Poole PJ, Rae B, Frankel A, Black PN. The effect of case management in patients with recurrent admissions for COPD (abstract). Am J Respir Crit Care Med 1999;159(3):A819.

59. Rich MW, Freedland KE. Effect of DRGs on three-month readmission rate of geriatric patients with congestive heart failure. Am J Public Health 1988;78(6):680–682.

60. Sander MH. Sleep heart health study: an update. 64th Annual Clinical Congress on Diseases of the Chest, American College of Chest Physicians. Toronto, Ontario, Canada. Nov, 1998.

61. Martens KH, Mellor SD. A study of the relationship between home care services and hospital readmission of patients with congestive heart failure. Home Healthc Nurse 1997;15(2):123–129.

62. Williams EI, Fitton F. Factors affecting early unplanned readmission of elderly patients to hospital. BMJ 1988;297(6651):784–787.

63. AARC Clinical Practice Guideline. Training the health-care professional for the role of patient and caregiver educator. Respir Care 1996,41(7):654–657.

64. Make B. Collaborative self-management strategies for patients with respiratory disease. Respir Care 1994,39(5):566–579.

65. Chervin RD, Theut S, Bassetti C, Aldrich MS. Compliance with nasal CPAP can be improved by simple interventions. Sleep 1997; 20(4):284–289.

66. Fletcher EC, Luckett RA. The effect of positive reinforcement on hourly compliance in nasal continuous positive airway pressure users with obstructive sleep apnea. Am Rev Respir Dis 1991,143(5 Pt 1):936–941.

67. Peckham DG, McGibbon K, Tonkinson J, Plumbley G, Pantin C. Improvement in patient compliance with long-term oxygen therapy following formal assessment with training. Respir Med 1998,92(10): 1203–1206.

68. Walshaw MJ, Lim R, Evans CC, Hind CR. Factors influencing the compliance of patients using oxygen concentrators for long term home oxygen therapy. Respir Med 1990,84(4):331–333.

69. Friedman M, Serby CW, Menjoge SS, Wilson JD, Hilleman DE, Witek TJ Jr. Pharmacoeconomic evaluation of a combination of

ipratropium plus albuterol compared with ipratropium alone and albuterol alone in COPD. Chest 1999;115(3):635–641.

70. Ikeda A, Nishimura K, Koyama H, Tsukino M, Mishima M, Izumi T. Dose response study of ipratropium bromide aerosol on maximum exercise performance in stable patients with chronic obstructive pulmonary disease. Thorax 1996;51(1):48–53.

71. Friedman M. Changing practices in COPD: a new pharmacologic treatment algorithm. Chest 1995;107(5 Suppl):194S–197S.

72. Shiner RJ, Zaretsky U, Mirali M, Benzaray S, Elad D. Evaluation of domiciliary long-term oxygen therapy with oxygen concentrators. Isr J Med Sci 1997;33(1):23–29.

73. Dale BA. Bacterial contamination of home nebulisers (letter). Br Med J (Clin Res Ed) 1987;295(6611):1486.

74. Ronchetti R, Indinnimeo L, Bonci E, Corrias A, Evans D, Hindi-Alexander M, et al. Asthma self-management programmes in a population of Italian children: a multicentre study. Italian Study Group on Asthma Self-Management Programmes. Eur Resp J 1997;10(6):1248–1253.

75. Salzman C. Medication compliance in the elderly. J Clinical Psychiatry 1995;56 Suppl 1:18–22, discussion 23.

76. Milgrom H, Bender B, Ackerson L, Bowry P, Smith B, Rand C. Noncompliance and treatment failure in children with asthma. J Allergy Clin Immunol 1996;98(6 Pt 1):1051–1057.

77. Pepin JL, Barjhoux CL, Deschaux C, Brambilla C. Long-term oxygen therapy at home: compliance with medical prescription and effective use of therapy. ANTADIR Working Group on Oxygen Therapy. Association Nationale de Traitement a Domicile des Insuffisants Respiratoires. Chest 1996;109(5):1144–1150.

78. Morrison D, Skwarski K, MacNee W. Review of the prescription of domiciliary long term oxygen therapy in Scotland. Thorax 1995; 50(10):1103–1105. Erratum in: Thorax 1995;50(12):1327.

79. Kampelmacher MJ, Van Kesteren RG, Alsbach GP, Melissant CF, Wynne HJ, Douze JM, Lammers JW. Prescription and usage of long-term oxygen therapy in patients with chronic obstructive pulmonary disease in The Netherlands. Respir Med 1999 Jan;93(1):46–51.

80. Ringbaek T, Lange P, Viskum K. Compliance with LTOT and consumption of mobile oxygen. Respir Med 1999 May;93(5):333–337.

81. Restrick LJ, Paul EA, Braid GM, Cullinan P, Moore-Gillon J, Wedzicha JA. Assessment and follow up of patients prescribed long term oxygen treatment. Thorax 1993;48(7):708–713.

82. American Association for Respiratory Care. Long-term oxygen therapy conference summary. Respir Care 2000;45(2):237–245.

83. Petty TL, Casaburi R. Recommendations of the Fifth Oxygen Consensus Conference. Respir Care 2000;45(8):957–961.

84. Kolbe J, Vamos M, Fergusson W, Elkind G. Determinants of management errors in acute severe asthma. Thorax 1998;53(1):14–20.

85. Siafakas NM, Bouros D. Consequences of poor compliance in chronic respiratory diseases. Eur Respir J 1992;5(1):134–136.

86. Long term domiciliary oxygen therapy in chronic hypoxic cor pulmonale complicating chronic bronchitis and emphysema. Report of the Medical Research Council Working Party. Lancet 1981;1(8222): 681–686.

87. Continuous or nocturnal oxygen therapy in hypoxemic chronic obstructive lung disease: a clinical trial. Nocturnal Oxygen Therapy Trial Group. Ann Int Med 1980;93(3):391–398.

88. Kampelmacher MJ, van Kestern RG, Alsbach GP, Melissant CF, Wynne HJ, Douze JM, Lammers JW. Characteristics and complaints of patients prescribed long-term oxygen therapy in The Netherlands. Respir Med 1998;92(1):70–75.

89. Medicare. Home Oxygen Program Warrants Continued HCFA Attention (Letter Report, 11/07/97, GAO/HEHS 98-17). Available at: http://www.gao.gov.

90. Problems in prescribing and supplying oxygen for Medicare patients. Am Rev Respir Dis 1986;134(2):340–341.

91. Further recommendations for prescribing, reimbursement, technology development, and research in long term oxygen therapy. Summary of the Fourth Oxygen Consensus Conference, Washington DC, October 15–16, 1993. Am J Respir Crit Care Med 1994 Sep; 150(3):875–7.

92. Petty TL, O'Donohue WJ Jr (Co-charimen). Conference report. New problems in supply, reimbursement, and certification of medical necessity for long-term oxygen therapy. Am Rev Respir Dis 1990;142:721–724.

93. Petty TL, O'Donohue WJ. Further recommendations for prescribing, reimbursement, technology development, and research in long-term oxygen therapy. Summary of the Fourth Oxygen Consensus Conference, Washington DC, October 15–16, 1993. Am J Respir Crit Care Med 1994;150(3):875–877.

94. Farrero E, Prats E, Maderal M, Giro E, Casolive V, Escarrabill J [Usefulness of home visits in the control and evaluation of the appropriate use of home continuous oxygen therapy.] Arch Bronconeumol 1998;34(8):374–378. Article in Spanish.

95. Morrison D, Skwarski KM, MacNee W. The adequacy of oxygenation in patients with hypoxic chronic obstructive pulmonary disease treated with long-term domiciliary oxygen. Respir Med 1997; 91(5):287–291. Erratum in Respir Med 1997;91(7):442.

96. Shwinski P, Lagosz M, Gorecka D, Zielinski J. The adequacy of oxygenation in COPD patients undergoing long-term oxygen therapy assessed by pulse oximetry at home. Eur Respir J 1994 7(2): 274–278.

97. Carroll N, Walshaw MJ, Evans CC, Hind CR. Nocturnal oxygen desaturation in patients using long-term oxygen therapy for chronic airflow limitation. Respir Med 1990;84(3):199–201.

98. Gorzelak K [Prognostic value of nocturnal pulse-oximetry in patients with chronic obstructive pulmonary disease disease treated with oxygen at home.] Pol Arch Med Wewn 1995;94(6):475–481 article in Polish.

99. Continuous or nocturnal oxygen therapy in hypoxemic chronic obstructive lung disease: a clinical trial. Nocturnal Oxygen Therapy Trial Group. Ann Intern Med 1980;93(3):391–398.

100. Stoller JK. Misallocation of respiratory care services: time for a change (editorial). Respir Care 1993;38(3):263–266.

101. Haney D, Kester L, Giles D, Komara JJ Jr, Fergus LC, Stoller JK. Early experience with the respiratory therapy consult service at the Cleveland Clinic Foundation (abstract). Respir Care 1992;37(11): 1282.

102. Browning JA, Kaiser DL, Durbin CG Jr. The effect of guidelines on appropriate use of arterial blood gas analysis in the intensive care unit. Respir Care 1989;34(4):269–276.

103. Hart SK, Dubbs W, Gil A, Myers-Judy M. The effects of therapist-evaluation of orders and interaction with physicians on the appropriateness of respiratory care. Respir Care 1989;34(3):185–190.

104. Kirby EG, Durbin CG Jr. Establishment of a respiratory assessment team is associated with decreased mortality in patients re-admitted to the ICU. Respir Care 1996;41(10):903–907.

105. Konschak MR, Binder A, Binder RE. Oxygen therapy utilization in a community hospital: use of a protocol to improve oxygen administration and preserve resources. Respir Care 1999;44(5):506–511.

106. Pilon CS, Leathley M, London R, McLean S, Phang PT, Priestly R, et al. Practice guideline for arterial blood gas measurement in the intensive care unit decreases numbers and increases appropriateness of tests. Crit Care Med 1997;25(8):1308–1313.

107. Shrake KL, Scaggs JE, England KR, Henkle JQ, Eagleton LE. A respiratory care assessment-treatment program: results of a retrospective study. Respir Care 1996;41(8):703–713.

108. Stoller JK, Mascha EJ, Kester L, Haney D. Randomized controlled trial of physician-directed versus respiratory therapy consult service-directed respiratory care to adult non-ICU inpatients. Am J Respir Crit Care Med 1998;158(4):1068–1075.

109. Torrington KG. Protocol driven respiratory therapy: closing in on appropriate utilization at comparable cost and patient outcomes. Chest 1996;110(2):313–314.

110. Brougher LI, Blackwelder AK, Grossman GD, Straton GW. Effectiveness of medical necessity guidelines in reducing cost of oxygen therapy. Chest 1986;90(5):646–648.

111. Stoller JK, Skibinski CI, Giles DK, Kester EL, Haney DJ. Physician-ordered respiratory care versus physician-ordered use of a respiratory therapy consult service: results of a prospective observational study. Chest 1996;110(2):422–429.

112. Borson S, Claypoole K, McDonald GJ. Depression and chronic obstructive pulmonary disease: treatment trials. Semin Clin Neuropsychiatry 1998 Apr;3(2):115–130.

113. Reisner C, et al. Depression, common and costly in chronic obstructive pulmonary disease (abstract). Chest 1998;114:341S–342S.

114. Reisner C, et al. Depression severity and smoking history, but not age or FEV₁, highly correlate with total charges and medical resource utilization in COPD (abstract). Chest 1998;114:341S.

115. Mahler DA. Pulmonary rehabilitation. Chest 1998 Apr;113(4 Suppl): 263S–268S.

116. Tiep BL. Disease management of COPD with pulmonary rehabilitation. Chest 1997;112(6):1630–1656.

117. Pulmonary rehabilitation: joint ACCP/AACVPR evidence-based guidelines. ACCP/AACVPR Pulmonary Rehabilitation Guidelines Panel. American College of Chest Physicians. American Association of Cardiovascular and Pulmonary Rehabilitation. Chest 1997; 112(5):1363–1396.

118. Ramsdell JW, Swart JA, Jackson JE, Renvall M. The yield of a home visit in the assessment of geriatric patients. J Am Geriatr Soc 1989;37(1):17–24.

119. Fabacher D, Josephson K, Pietruszka F, Linderborn K, Morley JE, Rubenstein LZ. An in-home preventive assessment program for independent older adults: a randomized controlled trial. J Am Geriatr Soc 1994;42(6):630–638.

120. Kravitz RL, Reuben DB, Davis JW, Mitchell A, Hemmerling K, Kington RS, Siu AL. Geriatric home assessment after hospital discharge. J Am Geriatr Soc 1994;42(12):1229–1234.

121. American Medical Association. Guidelines for physicians in the medical management of the home care patient, 1st ed. Chicago: AMA, 1992.

122. Zajac B. Respiratory wellness programs for MCO members with asthma and COPD (lecture). Presented at the American College of Healthcare Executives, 42nd Congress on Healthcare Management. Chicago IL: Mar 1999.

123. Warburton SW Jr, Futterman R, Rogheri JE, Skelly JJ. A respiratory disease management program for managed care (lecture). Presented at the NAEPP/ACCP National Conference on Asthma. Leesburg VA: Mar 1998.

124. Weber K, Harrisberger D, Hendon B. Impact of in-home intervention on measures of quality of life, functional status, and health-care utilization in patients with COPD. Eur Respir J 1998;12(Suppl 28):169S.

125. Hendon B, Kageler WV. Asthma and COPD disease management programs reduce healthcare utilization and improve quality of life (abstract). Respir Care 1996;41(10):957.

126. Rogheri JE, Shur J, Skelly JJ, Warburton SW. Asthma guideline compliance: impact of a disease management program (lecture). Presented at the NAEPP/ACCP National Conference on Asthma. Leesburg VA: Mar 1998.

127. Rogheri JE, Skelly JJ, Warburton SW. Respiratory Therapists as Care Managers in Asthma Disease Management. Presented at the NAEPP/ ACCP National Conference on Asthma. Leesburg VA: Mar 1998.

128. Cummings JE, Hughes SL, Weaver FM, Manheim LM, Conrad KJ, Nash K, et al. Cost-effectiveness of Veterans Administration hospital-based home care: a randomized clinical trial. Arch Intern Med 1990;150(6):1274–1280.

129. Dranove D. An empirical study of a hospital-based home care program. Inquiry 1985 Spring;22(1):59–66.

130. House Report No. 100-391(II), accompanying HR 3545, page 392. Available at: http://www.house.gov/

131. Kribbs NB, Pack AI, Kline LR, Smith PL, Schwartz AR, Schubert NM, et al. Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. Am Rev Respir Dis 1993; 147(4):887–895.

Letters

Lung Collapse During Low Tidal Volume Ventilation in Acute Respiratory Distress Syndrome

Kallet et al recently reported a case of acute respiratory distress syndrome complicated by retained secretions and lung collapse.[1] The authors offer a good discussion about the possible effects of a low tidal volume (V_T) ventilatory strategy on bronchial hygiene. I think most clinicians would concede that they focus more of their attention (a finite commodity) on plateau pressure, positive end-expiratory pressure, and oxygenation indices than on bronchial hygiene when assessing an ARDS patient. I would like to add to the discussion by commenting on the use of small V_T in acute lung injury/acute respiratory distress syndrome patients with decreased chest wall compliance, like the authors' patient with increased abdominal pressure. Though it is enticing to use plateau pressure as a reflection of alveolar distention, one must always consider the contribution of chest wall elastance to plateau pressure, since plateau pressure is generated by the elastic state of the total respiratory system. When plateau pressure is increased because of decreased chest wall compliance, lowering already conservative V_T to avoid high plateau pressure can result in atelectasis, deoxygenation, and perhaps as the authors suggest, retained secretions as a consequence of unopposed increases in pleural pressure.[1,2] Lowering V_T may be a mistake, not merely a necessary evil of providing protective ventilation, since using a V_T that produces plateau pressures above "protective" limits (35–40 cm H_2O) in the presence of decreased chest wall compliance does not necessarily produce ventilator-induced lung injury. Dreyfuss et al[3] showed that when mechanically ventilated rats were exposed to high airway pressures because a strap was placed around the thorax, ventilator-induced lung injury does not occur. I agree with the authors' suggestion that bronchial hygiene measures should be given more attention when using a low V_T ventilatory strategy. However, we must also be mindful not to unnecessarily lower V_T at the behest of a potentially misleading ventilatory target. Until we have better methods of measuring transpulmonary pressure, we must rely on global respiratory system measurements, which need to be interpreted cautiously when assessing lung mechanics.

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REFERENCES

1. Kallet RH, Siobal MS, Alonso JA, Warnecke EL, Katz JA, Marks JD. Lung collapse during low tidal volume ventilation in acute respiratory distress syndrome. Respir Care 2001;46(1):49–52.
2. Mutoh T, Lamm WJE, Embree LJ, Hildebrandt J, Albert RK. Abdominal distension alters regional pleural pressures and chest wall mechanics in pigs in vivo. J Appl Physiol 1991;70(6):2611–2618.
3. Dreyfuss D, Soler P, Basset G, Saumon G. High inflation pressure pulmonary edema. Respective effects of high airway pressure, high tidal volume, and positive end-expiratory pressure. Am Rev Respir Dis 1988;137(5):1159–1164.

The author responds:

Mr Haynes raises a valid point when he questions the need for radical tidal volume (V_T) reduction in patients with acute respiratory distress syndrome (ARDS) and reduced chest wall compliance (C_{CW}). Early on in the development of lung-protective ventilation in ARDS, a target end-inspiratory plateau pressure (P_{PLAT}) of 35 cm H_2O was chosen because under conditions of normal lung-thorax mechanics, the lungs generally reach total capacity at that transpulmonary pressure.[1] Later on, the target P_{PLAT} range was reduced to 25–30 cm H_2O because further evidence suggested that lung injury may occur at lower pressures.[2] Therefore, it has been assumed implicitly that, in ARDS, C_{CW} is normal (152–285 mL/cm H_2O).[3,4] As Mr Haynes correctly observed, animal models of ARDS have demonstrated that when *thoracoabdominal* strapping is used to reduce C_{CW}, lung injury does not occur at the expected levels of P_{PLAT}, thus giving birth to the concept of "volutrauma"

as an important factor in ventilator-induced lung injury.[5] In fact, several studies[6,7] have reported markedly reduced C_{CW} (75–137 mL/cm H_2O) in ARDS patients. Therefore, it would appear that our attempts to impose radical V_T reduction (4–5 mL/kg) to achieve a desired P_{PLAT} may be unwarranted.

However, this situation may not be as straightforward as Mr Haynes suggests. First, the mechanical properties of the chest wall are complex and do not conform to a simplistic model of a single elastic structure coupled to the lungs. The chest wall comprises both the rib cage and the abdominal wall, each with a distinct compliance. Furthermore, additional complexities are added when body posture changes or when large applied forces distort the surface shapes.[8] In the supine position during spontaneous breathing, the rib cage contributes only 30% to tidal ventilation.[9] In contrast, during controlled mechanical ventilation at the same V_T, the rib cage accounts for over 70% of displacement, as the lower abdominal wall compliance becomes apparent when it is passively displaced.[9] In the supine position, small V_T ventilation and the volume-enhancing effects of positive end-expiratory pressure are preferentially distributed to the ventral portions of the lung.[10] This discrepancy between rib cage and abdominal wall compliance is greatly magnified under conditions of intra-abdominal hypertension, when the abdominal contents protrude into the thoracic cavity and alter intrathoracic pressures.[11] When I have observed chest excursions during controlled ventilation in these patients, often only the upper chest appears to be displaced. Therefore, it is possible that what would constitute a lung-protective V_T (6–7 mL/kg) in patients with normal C_{CW} may cause regional overdistention and ventilator-induced lung injury in a patient with abdominal compartment syndrome. The fact that this apparently did not happen when the V_T was increased from 4.5 to 7 mL/kg in our case does not preclude the possibility. When it appears that C_{CW} is globally reduced (ie, because of generalized tissue edema from sepsis) or from muscular rigidity due to fentanyl administration), then I would agree with Mr Haynes that limiting P_{PLAT} assumes less importance. However, I would urge caution in deviating

from lung-protective ventilation goals of $P_{PLAT} \leq 30$ cm H_2O when diminished C_{CW} occurs because of elevated intra-abdominal pressure.

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REFERENCES

1. Marcy TW, Marini JJ. Inverse ratio ventilation in ARDS: rationale and implementation. Chest 1991;100(2):494–504

2. Tsuno K, Prato P, Kolobow T. Acute lung injury from mechanical ventilation at moderately high airway pressures. J Appl Physiol 1990;69(3):956–961.

3. Grimby G, Hedenstierna G, Lofstrom B. Chest wall mechanics during artificial ventilation. J Appl Physiol 1975;38(4):576–580.

4. Van Lith P, Johnson FN, Sharp JT. Respiratory elastances in relaxed and paralyzed states in normal and abnormal men. J Appl Physiol 1967;23(4):475–486.

5. Dreyfuss D, Soler P, Basset G, Saumon G. High inflation pressure pulmonary edema: respective effects of high airway pressure, high tidal volume, and positive end-expiratory pressure. Am Rev Respir Dis 1988; 137(5):1159–1164.

6. Katz JA, Zinn SE, Ozanne GM, Fairley HB. Pulmonary, chest wall, and lung-thorax elastances in acute respiratory failure. Chest 1981;80(3):304–311.

7. Conti G, Vilardi V, Rocco M, DeBlasi RA, Lappa A, Bufi M, et al. Paralysis has no effect on chest wall and respiratory system mechanics of mechanically ventilated, sedated patients. Intensive Care Med 1995; 21(10):808–812.

8. Kallet RH, Campbell AR, Alonso JA, Morabito DJ, Mackersie RC. The effects of pressure control versus volume control assisted ventilation on patient work of breathing in acute lung injury and acute respiratory distress syndrome. Respir Care 2000;45(9): 1085–1096.

9. Smith JC, Loring SH. Passive mechanical properties of the chest wall. In: Fishman AP, Macklem PT, Mead J, Geiger SR, editors. Handbook of physiology. Section 3—The respiratory system. Vol III: Mechanics of breathing. Chapter 25. Bethesda: American Physiologic Society; 1986: 429–442.

10. Bindslev L, Hedenstierna G, Santesson J, Norlander O, Gram I. Airway closure during anaesthesia and its prevention by positive end expiratory pressure. Acta Anaesthesiol Scand 1980;24(3):199–205.

11. Cullen DJ, Coyle JP, Teplick R, Long MC. Cardiovascular, pulmonary, and renal effects of massively increased intra-abdominal pressure in critically ill patients. Crit Care Med 1989;17(2):118–121.

12. Nuytinck JK, Goris RJ, Weerts JG, Schillings PH, Stekhoven JH. Acute generalized microvascular injury by activated complement and hypoxia: the basis of adult respiratory distress syndrome and multiple organ failure? Br J Exp Pathol 1986;67(4): 537–548.

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Irwin and Rippe's Intensive Care Medicine, 4th edition (2 volume set). Richard S Irwin MD, Frank B Cerra MD, James M Rippe MD, Editors. Philadelphia: Lippincott Raven. 1999. Hardcover, illustrated, 2,519 pages, $225.

Now in its fourth edition, **Irwin and Rippe's Intensive Care Medicine** has become one of the most popular critical care reference books in the United States. The editors' stated goal is to create a book with a multidisciplinary approach to critical care. Though the scope has broadened in this edition, the editors wanted to preserve a practical and clinical approach to the critically ill. They have achieved their goals and created an impressive reference tool that is the most complete of all the major critical care textbooks.

This is a thick two-volume textbook that addresses all aspects of intensive care medicine. The books come in handsome blue covers. The paper is a bit thin and fragile, but the binding held up well to wear and tear. The text was easy to read and the editing was excellent, with only a few typographical errors noted.

The editors have assembled over 300 authors to write 227 chapters grouped into 18 sections. The authors are for the most part experts in their fields, although there is a clear bias toward physicians from the editors' home institutions. The organization is traditional, based on medical specialty and organ system. There are also sections on intensive care unit (ICU) procedures, pharmacology/poisonings, surgical problems, shock, transplantation, nutrition, and a section on moral, ethical, and legal issues in intensive care. The sections are quite thorough, with multiple chapters in each, usually organized by diagnosis. The chapters are all quite detailed and extensively referenced.

The traditional sections include cardiology, pulmonary, renal, gastroenterology, infectious diseases, endocrine, hematology, neurology, psychiatry, and rheumatology. The most complete of these sections were the pulmonary, psychiatry, and endocrine sections. In the pulmonary section there could have been more information on mechanical ventilation, but the other pulmonary chapters were excellent, especially the

chapters on basic physiology and extrapulmonary causes of respiratory failure. The endocrine section contained detailed information on numerous subjects that are often misunderstood by intensivists, such as thyroid disease and mineral metabolism. I would have liked to see more information on controversial areas such as relative adrenal insufficiency in the ICU and the use of growth hormone.

The cardiology parts of this textbook are split into two sections: one on cardiovascular problems in the ICU and the other on coronary care. I found the separation a bit arbitrary, with valvular disease covered in one section and heart failure and ischemia covered in the other. Overall, this section was one of the weaker parts of the textbook, often filled with more dogmatic statements rather than recommendations based on evidence (eg, volume loading in right ventricular infarction).

The section on gastroenterology was well written but less extensive than the other sections and probably deserved more detailed coverage. The renal, hematology, infectious disease, neurology, and rheumatology sections were all well written and covered a broad range of relevant topics.

The other sections of this textbook were innovative and covered information often not available in other medical critical care books. The section on procedures and monitoring is one of the highlights of this textbook. This section devotes over 280 pages to monitoring and procedures ranging from arterial lines to percutaneous bladder tube placement. The figures and the technical aspects of this section are excellent.

The sections on echocardiography, chest tube placement, and tracheotomy are particularly good. An innovative section on invasive radiologic procedures was very useful. My only problem with this section was that some of the conclusions made on interpretation of data (eg, pulmonary arterial catheters) were a bit superficial and in some cases based on dogma rather than strong support in the literature.

The pharmacology and poisoning section was excellent, with informative chapters on kinetics followed by a great chapter on the general approach to the poisoned patient.

The section then covered an extensive list of common poisonings.

The surgical, transplant, nutrition, and shock/trauma sections were very good, although there were some areas not addressed (eg, the role of enteral nutrition in acute pancreatitis). Every chapter is very well referenced, which makes further research on topics not fully covered in the text very easy.

The scope of this textbook is impressive and each topic addressed is thoroughly covered. In general, the writing is excellent and well organized. There are some holes in the coverage, but that is expected in a book covering a field as broad as intensive care medicine. Compared to similar textbooks, this is the most broad-based in its applicability and is clearly the textbook to be shelved in every ICU library. However, its organization assumes a fair amount of familiarity with the subject matter, which may limit the use of the textbook to those more experienced in the field. Critical care practitioners would probably want to own this book and use it as the definitive reference in caring for the critically ill. It is too detailed to be read cover to cover by fellows studying for the boards, and likewise may be unnecessarily complete for residents or critical care nurses. Respiratory therapists, clinical nurse specialists in critical care, and critical care fellows will definitely want access to it while in the ICU. Overall I think the authors have created the definitive textbook for care of the critically ill patient and should be commended on their effort.

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Procedures and Techniques in Intensive Care Medicine, 2nd edition. Richard S Irwin MD, James M Rippe MD, Frank B Cerra MD, Frederick J Curley MD, and Stephen O Heard MD, Editors. Philadelphia: Lippincott Williams & Wilkins. 1999. Softcover, illustrated. 307 pages plus index. $59.95.

This is an ambitious, multidisciplinary book whose aim is the explication and teaching of almost all procedures done in the intensive care setting. It is published both as

a stand-alone volume and as the first section of a full-scale critical care text edited by the same authors. Though the authors suggest a wide target readership (ranging from surgeons to emergency medicine physicians and nurses), the presentation appears more attuned to the needs of house staff rotating on an intensive care service: broad-based, not too detailed, focused on skills they will actually use. Nurses and respiratory therapists will probably find the level of detail provided about setup, care, and use of the described equipment insufficient to guide day-to-day practice.

The stand-alone paperback book, presumably offered as a lower-cost alternative to the full textbook, compromises pocket-ability in favor of full-size illustrations. The typesetting is professional: the text is crisp, clear, and legible. The illustrations are black-and-white, but of uneven quality. The majority of the illustrations were done by a K Powell, and they boast clean outlines, subtle shading, and sufficient detail to guide the practitioner—they are excellent. Other illustrations and some photographs, however, appear to have been digitally scanned, with disappointing results: the photograph of a pacemaker (page 74), for example, has no legible buttons.

The book lacks internal structure, most likely a result of its origin as a part of a larger text. Each procedure is discussed in a separate chapter. There is no formal grouping into sections or systems, but there seems to be a general progression from line placements through cardiac to pulmonary, gastrointestinal, and neurologic/neurosurgical procedures. Some procedures on the list seem less than "critical"—joint aspiration, for example—whereas others are oddly absent: cardiopulmonary resuscitation and advanced cardiac life support are only briefly mentioned in the chapter on defibrillation, and the technique of intraosseous bloodstream access is not discussed at all. The book is focused on the care of adults; pediatric-specific issues are covered poorly if at all. Another consequence of the book's origin within a full-size text is the overwhelming number of references. There are 194 references in the chapter on placement of a pulmonary artery catheter—far more than are needed to understand the processes of insertion and troubleshooting.

The wide variety of procedures performed in the intensive care setting leads to great variance in the amount of coverage offered to each. Arterial puncture, for ex-

ample, receives a 4-page chapter quite apart from the 10 pages already devoted to placement and care of arterial catheters, whereas the exceedingly complex and specialized topic of temporary mechanical assistance for left ventricular failure receives a scant 8 pages of discussion. The result is that the book cannot be relied on to successfully troubleshoot the more complex devices, though it does provide a sound introduction to their operating principles.

The text offers several inviting high points. Percutaneous tracheostomy receives detailed attention and excellent photographic illustration. The endoscopically guided placement of feeding tubes is well described. The review of dialysis contains an excellent discussion of dialysis theory, simplifying this complex subject as well as providing an excellent practical guide. The mechanics of central and arterial line placement and chest tube placement are also well described.

On the other hand, some chapters simply lack critical care "common sense." The opening chapter, on endotracheal intubation and airway management, contains this astounding statement: "Since patients requiring intubation often have a depressed level of consciousness, anesthesia is usually not required." These authors clearly do not routinely work in the intensive care setting! The chapter on neurologic monitoring offers an illustration of the placement of leads for somatosensory evoked potentials—which would generally only be placed by a specially trained technician—but neglects to provide an image or explanation of a ventriculostomy drain setup, perhaps the most commonly used monitor the average nurse or house staff officer will see.

This second edition text also suffers from a certain complacency. Some chapters that slipped through largely unaltered from the first edition should have been more heavily revised. Some techniques described are simply outdated. Catheter-through-needle thoracentesis kits, though described eloquently in this text, have been superseded by catheter-over-needle kits, which eliminate the risk of shearing off the catheter. Glass syringes for arterial blood gas samples are museum pieces. Other newer techniques are left out. Noninvasive ventilation, a rapidly growing modality of ventilatory support, receives no mention whatsoever. Likewise, discussion of ultrasound guidance for central venous catheter placement—a clear advance in ease and safety of performing these

procedures—is absent. Diagnostic peritoneal lavage is covered; measurement of abdominal compartment pressure is not.

This book is caught in an awkward adolescence. It spent a happy childhood as a practical procedure manual for a number of basic intensive care unit procedures. These chapters are well written, if a bit dated, and detailed enough to be the sole reference of a house staff officer contemplating a late-night thoracentesis. Having discovered wider horizons—the roles of endoscopy, bronchoscopy, dialysis, and advanced techniques of circulatory support—it is struggling to keep up with the hip kids down the street. The chapters dealing with these topics are current at the price of incorporating less detail. They would be of more use for discussing the merits of a procedure on rounds than actually performing it. Still, the authors are to be commended for their effort: concise coverage of these topics in an easily accessible book is in itself a worthwhile goal.

Overall, this book is well suited to the needs of a fairly narrow target audience: house staff officers rotating through a general intensive care unit. Nurses and respiratory therapists will find it helpful in explaining some uncommon procedures. It would be a worthwhile addition to the general reference shelf in any break room.

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Handbook of Pediatric Intensive Care, 3rd edition. Mark C Rogers MD and Mark A Helfaer MD, Editors. Philadelphia: Lippincott Williams & Wilkins. 1999. Softcover, illustrated, 994 pages, $49.95.

The third edition of **Handbook of Pediatric Intensive Care**, edited by Mark Rogers and Mark Helfaer, represents a change in overall focus. Previous editions seemed to be designed to limit detail and give more of a "what you see, how you treat it" approach, whereas this edition seems to greatly expand on detail provided and to focus less on easy-to-use algorithms. The increase in information provided makes the handbook overall a more complete reference than earlier editions. The down side is that with the increase in content comes a large increase in size and weight. This handbook is impossible to fit in a coat pocket or to carry around easily. This may make it less desir-

able as a reference that is easily portable and pocket-sized. In addition, the depth of information provided in the handbook compared to the full text makes it almost not worth having both. Conversely, the advantage of having a more complete handbook at a smaller size and cost than the full text is a distinct advantage in many circumstances.

The chapters are outlined in a format that allows quick identification of specific topics and where to find data regarding specific disease states, treatment algorithms, and basic information. The tables suffer from lack of presentation to make them easier to read, but are plentiful and useful in content. The pediatric drug dosage guide in the appendix and the various commonly used formulas are very helpful to medical personnel at all levels of training and expertise.

The first few chapters deal with common intensive care unit subjects in terms of basic cardiopulmonary resuscitation and airway management. Though the majority of the information is available in other texts and is well known to most critical care providers, a good overview of these subjects is provided. In a similar fashion, the basic overall management of bronchiolitis and asthma is presented at a level appropriate for multiple specialties and levels of expertise. I found regrettable the omission of discussion of intravenous terbutaline for asthma and the uncommon but potentially life-saving role of extracorporeal life support in these disease states.

One of the major disappointments in the book was the chapter on acute respiratory distress syndrome. The coverage was very brief and little detail was given in regard to the observed decline in mortality from this disease. The general algorithm that outlined management guidelines for acute respiratory distress syndrome was useful. The chapter regarding respiratory support and mechanical ventilation went in the other direction—very comprehensive for a "handbook." The discussion of high-frequency ventilation was lacking in detail, however, and a table outlining general setup and use of this modality for patients of different ages and sizes would be of interest to those who use this modality infrequently.

There were many chapters that covered disease states that are not frequently included in a general handbook. Although these were often much more comprehensive than perhaps needed, the sections on neuromuscular

disease and encephalopathies were worthwhile.

The cardiac sections were extremely well done. Although the graphics of what cardiac surgical repairs entail add pages to the book, they are extremely useful in understanding what procedures are performed in the operating room. Similarly, the graphics of electrocardiograph abnormalities and what to look for are very useful.

In a similar fashion, the coverage of head and spinal cord injury and central nervous system subjects such as meningitis were very good. A related topic, brain death, was also well done, with good detail. The practical aspects of care to successfully protect potential donor organs to the time of harvest was very well done. This is an important topic that is rarely discussed.

The remainder of the book was also fairly comprehensive and covered most of what is found in critical care. The only specific population that seemed lacking was oncology patients—an important and difficult population in critical care, especially as more rigorous chemotherapy regimens lead to secondary complications and the need for intensive care.

For medical personnel who are interested in critical care, this text provides a good overall reference. As a general reference for your personal library, the full Rogers *Textbook of Pediatric Intensive Care* may be more useful and easier to read. As a book that can be carried along in a backpack, the handbook will prove superior to the full textbook. Choosing which is best depends on the specific needs and desires of the consumer.

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Drugs in Anaesthetic and Intensive Care Practice, 8th edition. MD Vickers MB BS DA(Eng), M Morgan MB BS DA(Eng), PSJ Spencer PhD DSc, MS Read MB BS. Oxford: Butterworth Heinemann. 1999. Hardcover, illustrated, 526 pages. $55.

Since the first edition appeared in 1962, this book has, over a period of 37 years, proven itself to be one of the most important references to drugs used in anesthetic practice, by having a record of publishing 8 editions and 5 reprints.

This eighth edition is both an expansion and comprehensive revision of the 1991 work. There are many changes in this edition. The phrase "intensive care" has been added to the book's title, to become "Drugs in Anaesthetic and Intensive Care Practice." The reason for that change is the awareness of a current development in intensive care, which is "a clear requirement of trainees in anesthesia, with the evidence that the great majority of intensive care units in the United Kingdom are managed and generally staffed by anaesthetists." The traditional title would imply too narrow a focus in this modern time. Consequent to the change, a new author, Dr Martyn Read, has been recruited. Naturally, the book's coverage has been widened to include drugs applied in the intensive care unit. All the existing chapters have been updated and many have been reorganized completely. Another addendum to this edition is the inclusion of synonymous drug names. When presenting a drug that has a different name in the United Kingdom than in the United States, the authors use the United States name or the International Nonproprietary Name and give the British Approved Name in parentheses on first mention within a section. The book now contains 526 pages and is an essential source of reference for all those involved in pharmacology.

The primary audience of this book is trainees in anesthesia, who should find the book an authoritative reference to the drugs they use in daily practice and an essential aid in preparing for fellowship examination. The book is also an excellent reference source for physicians when unusual situations occur. Moreover, it is also of value to respiratory therapists and nurses, as their daily practice also requires knowledge of many of the drugs discussed in the text.

Overall, this book is clearly organized and well written. It achieves its aim of succinctly reviewing most of the drugs used in modern anesthesia and intensive care practice. The first chapter ("General Pharmacology") and the 16th chapter ("Chemical Transmitters and Enzymes") both give an overview of pharmacology information that is succinct and up-to-date. There is one chapter (Chapter 20) that deals with infusion fluids and oxygen-carrying solutions. Each of the other 17 chapters describes different groups of drugs. The groupings are under different topics, such as therapeutic strategy (eg, bronchodilation), groups of receptors (eg, β-adrenergic agonists and antagonists),

common chemical features (eg, benzodiazepines), and action on the same organ or system (eg, heart, circulation, uterus, or endocrine glands). The authors devoted the beginning portion of each chapter to a review of the pharmacologic basis of that particular group of drugs or therapy subject. Monographs on different drugs or components of that group and notes on similar compounds that exhibit minor variations then follow.

One minor criticism of the book is that not enough emphasis has been placed on unifying ways of presenting material among authors; thus, the format of the book suffers a lack of uniformity:

1. When a drug is discussed in a separate section, some authors give the page number where the material was previously discussed, whereas others do not. For example, in the review of haloperidol and droperidol, different materials concerning the two drugs have been separately mentioned in the beginning portion and the monograph. However, there is no indication in the monograph to inform readers about the previous material. Readers who are only reading the monographs for reference would miss important materials in the earlier section.

2. Of the 20 chapters in the book, only 3 chapters (1, 3, and 16) included references. Reference-free material makes the book slimmer and easier to read. However, it lacks evidence base and fails to facilitate the acquisition of further information.

The strength of this book is that it links clinical conditions to pharmacology. There are many useful summary tables and illustrations, which add substantially to the value of the text. The weakness of the book stems from the authors' attempts to have an encyclopedic coverage, giving little in-depth discussion to each individual entity.

The external appearance of the book is appealing, and it is the size of most standard textbooks. The appendix provides quick reference tables to new recommended drug nomenclature for the United Kingdom, though that would not be relevant to readers in the United States. The index appears useful and appropriate.

In summary, the authors have provided an extremely concise, useful, thorough, up-to-date review of drugs in the context of modern anaesthesia and intensive care practice. It is an excellent reference book for medical students and residents who have already had or are currently taking courses in anesthesia and critical care. Respiratory

therapists will definitely benefit from this book.

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The Haemodynamic Effects of Nitric Oxide, Robert T Mathie and Tudor M Griffith, Editors. London: Imperial College Press. 1999. Hardcover, illustrated, 518 pages. United Kingdom £55, United States $90.

Nitric oxide (NO) was identified as endothelium-derived relaxing factor in 1987. In the subsequent years, our knowledge of the biological roles of NO has exponentially grown, with the publication of over 35,000 articles. Although there have been many focused review articles attempting to summarize portions of this literature, this book represents one of the first attempts to provide a comprehensive overview of the area with an emphasis on the cardiovascular effects of NO. The chapters are written by recognized international NO experts, with only one third of the authors being from the United States.

The book is divided into 3 sections: physiology and biochemistry of NO, peripheral vascular effects of NO, and clinical implications of NO. The first section consists of 11 chapters, which focus on the chemistry, biosynthesis, and metabolism of NO. These chapters are exceptionally well written and present a complex mass of data in an understandable fashion. The use of well designed figures adds to the comprehensibility of this potentially confusing subject. This section of the book will be of definite value to anyone involved in NO-related research, and I have frequently used it as a reference volume for manuscripts and grants.

The second section of the book consists of 7 chapters that examine the effects of NO in regional circulations, including the brain, heart, skeletal muscle, liver, intestines, and kidney. These chapters present a balanced view of the contradictory effects of NO in many organs. Again, the information is presented in a complete manner, and the authors attempt to reconcile contradictory data whenever possible.

The third section of the book consists of 6 chapters and examines the effects of NO in ischemia-reperfusion injury, atherosclerosis, endothelial dysfunction, the pulmonary circulation, systemic hypertension, and septic shock.

In general, the chapters are comprehensive and well-written. In contrast to many textbooks in which the information in different chapters is contradictory, this book avoids that problem and is edited to emphasize the interrelationships of the chapters. The chapters are written at a state-of-the-art level that can sometimes be challenging for the casual reader. The majority of the chapters have over 100 references, so that this book could have been a single source for almost all information on NO that would be needed by a researcher or clinician.

Unfortunately, the book has two important limitations in this regard. First, although the expressed purpose of the book is to provide a state-of-the-art understanding of NO, most of the chapters are already substantially outdated. The overwhelming majority of references are from the period of 1992 to 1996, when a detailed understanding of NO was just beginning to be achieved. There are few references from 1997 and almost no references from 1998. Thus, the chapter on septic shock refers to the 1996 clinical trials of tumor necrosis factor (TNF-α), has no references to the use of selective inducible nitric oxide synthase (iNOS) inhibitors after 1995, and only refers to an abstract on the beneficial hemodynamic effects of N-monomethyl-L-arginine (L-NMA); the final results of the randomized clinical trial indicated increased mortality with L-NMA. Similarly, the discussion of the effects of NO in acute respiratory distress syndrome does not include any of the multiple clinical trials that focused on outcome; several of these studies were presented at meetings in 1997 and the phase 2 randomized study was reported by Dellinger et al in January 1998.[1] Thus, this book, which was published in September 1999, no longer provides an up-to-date source of information. It may be unavoidable that a book will be outdated shortly after its publication; unfortunately, this is a major problem in a field such as NO, which is rapidly evolving.

The second major limitation of the book (at least for many readers of RESPIRATORY CARE) is that the emphasis on basic science and laboratory research data limits the book's coverage of clinical issues. For example, I anticipated that there would be extensive coverage of inhaled NO. However, there are only two paragraphs on the effects of inhaled NO in acute respiratory distress syndrome, one paragraph on inhaled NO and persistent pulmonary hypertension of the newborn, and one paragraph on inhaled

NO and primary pulmonary hypertension. In the chapter on the role of NOS inhibition in septic shock, there are 7 paragraphs on animal studies and only 2 paragraphs on human studies. The chapter on the role of NO in ischemia-reperfusion injury is excellent but refers to no clinical studies.

In the past 2 years, over 10 books on NO have been published, each of which provides an excellent overview of the field. **The Haemodynamic Effects of Nitric Oxide** distinguishes itself by providing a comprehensive but understandable approach that will primarily be of value to researchers in this important subject.

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REFERENCE

1 Effects of inhaled nitric oxide in patients with acute respiratory distress syndrome: results of a randomized phase II trial. Inhaled Nitric Oxide in ARDS Study Group. Crit Care Med 1998;26(1):15–23

Thoracic Anaesthesia: Principles and Practice. S Ghosh BSc MBBS and RD Latimer MA MBBS. Oxford: Butterworth Heinemann. 1999. Hardcover, illustrated, 335 pages. $85.

Thoracic surgery has evolved from a high-risk, high-mortality attempt to save life into a highly specialized field directed toward the diagnosis and treatment of many intrathoracic conditions. The mortality rate associated with thoracic surgery has continued to decrease, despite the fact that surgeons are operating on older, sicker patients. Thoracic surgery presents a unique set of challenges to the anesthesiologist entrusted with the care of these compromised patients: not only must they ensure patient safety, they must also optimize the surgical field. The challenges include the care of a patient whose underlying pulmonary compromise leaves little room for error, in the face of their physiologic derangement and the need for one-lung ventilation. Anesthesia for thoracic surgery encompasses a broad spectrum of topics, including the physiologic, anatomic, pharmacologic, and clinical considerations for the patient undergoing pulmonary and esophageal surgery.

Thoracic Anaesthesia: Principles and Practice does an admirable job of attempting to cover the broad spectrum of thoracic anesthesia in an easily read, interesting, and informative volume. The text is written by many different authors, each of whom brings expertise and experience to different sections of the book, which covers the whole scope of thoracic anesthesia. Each chapter addresses a specific topic and is complete in itself. The text is directed at the anesthesia care provider who will be administering anesthetic to patients undergoing thoracic surgery.

This book is not only directed toward the anesthesia novice, but also to the senior anesthesiologist who would like to refresh his or her knowledge or brush up to take an examination. The anesthesia resident who will be taking care of these patients for the first time and who needs a thorough background in the anatomy, physiology, and procedures will benefit from the information contained in this text. An in-depth description of the surgical procedures, their indications and contraindications, as well as pitfalls and common complications associated with the procedures are addressed. This text is also useful for the senior anesthesia care provider, who may use it as an aide-memoir or for teaching purposes. The use of table summaries at the end of each chapter makes this a valuable book for review.

The initial chapters deal with the historical perspective of thoracic surgery and how the field of anesthesia developed from a risky, high-mortality pursuit to the modern, relatively safe administration of anesthesia with improved equipment and safety measures. It gives useful background information to the reader interested in the evolution of this field and what challenges prompted the development of much of the equipment in use today. The use of illustrations and photographs enhances this chapter and gives an appreciation of the obstacles presented to those early pioneers in the field and how they overcame them. Undoubtedly the equipment in use today provides patients with a safer and gentler anesthesia than was administered in the past.

A discussion of the physiology of one-lung ventilation follows, with particular focus on the effects of anesthesia and paralysis. This chapter will be useful to all anesthesia care providers, nurses, and respiratory therapists who care for these patients intra-operatively and post-operatively in the intensive care unit. The author of this chapter writes for the medically trained practitioner who already has a working knowledge of pulmonary physiology.

A very brief and superficial chapter on the anatomy of the lungs, with many diagrams (not always related to the text), completes the first part of the book. The indications and techniques for one-lung surgery are discussed in detail, with a focus on pitfalls, complications, and approach to treatment of these known complications. The chapter is thorough and logically written and provides many useful tips to avoid pitfalls for the inexperienced anesthesia care provider caring for these patients for the first time.

The following chapters (5–9) focus on the specific operations carried out on the lungs, pleura, diaphragm, airway, and esophagus. The authors have thoroughly researched each disease process, the surgical procedure, and the anesthesia care these patients require. An in-depth review of the pre-operative, intra-operative, and post-operative complications that can occur in this patient population is provided for each specific procedure and how it should be diagnosed and treated. These chapters will be of use to any health care provider who cares for these patients before, during, and after their surgery. They are especially helpful to the anesthesia care provider who will be intimately involved with the administration of the anesthetic during the surgical procedure. The intensive care unit staff who care for these patients post-operatively will benefit from a working knowledge of what occurred intra-operatively, as this will lead to better post-operative care and anticipation of expected complications.

A discussion on newer techniques, such as video-assisted thoracoscopic surgery and lung volume reduction surgery, is included and discussed in detail. This section will really only be of interest to the operating room anesthesiologist, as none of these procedures occur out of the operating room.

The field of pediatric thoracic anesthesia is addressed in the next chapter, with a description of much of the childhood pathology that warrants such anesthesia. This topic can easily be discussed as a textbook in its own right, and this chapter makes an admirable attempt to cover this vast topic in a short space, but lacks the depth of information required to really take care of these children undergoing thoracic anesthesia. The specific workup of these children is directed to the requirements of the British

medical system and may be quite different in the American medical environment.

The final chapters of this book deal with post-operative pain relief, commonly seen post-operative complications and modes of respiratory support, as well as the controversies in the field between the different schools of thought. This final chapter shows that experts in their fields have opinions that differ from one another. It gives the novice an insight into an evolving and advancing field, while allowing one the leeway to develop one's own anesthesia practice within the guidelines recommended in this book.

The book is relatively short, 335 pages, and light enough to carry around. It is well laid out and has an easy-to-read style, with good use of pictures, tables, and graphics. I

think that the summaries at the end of most of the chapters will be helpful to those using this book as a study aid. The information is factual, informative and accurate. The index is useful for quick reference to the key concepts put forward in the book.

In summary, I feel that the editors achieved their stated goal of providing a useful text for the benefit of junior and senior anesthesiologists administering anesthesia for thoracic surgery. The terminology is written in the English style with the use of "theatre" for "operating room" and "high dependence area" for "intensive care unit," which in no way detracts from the valuable information that it imparts to the reader. I do not think this is a book that would be of primary interest to the respiratory therapist or bedside nurse, as it is mostly

directed at pre-operative and intra-operative care, though some of the chapters may provide insight into a patient's post-operative course. Very little post-operative information is given for these patients as, on the whole, anesthesiologists are not responsible for the post-operative care of these patients, which falls back into the realm of the attending thoracic surgeon.

I, as an anesthesiologist, enjoyed reading this book.

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CORRECTION

In the letter "Aerosols and the Profession of Respiratory Care:
Leading the Way Out of the Fog" by Kenneth E Noblett (Respir Care
2001;46[3]:275-276) the location of the author is in error. He is from
Evansville, Indiana — not Illinois.

The American Association for Respiratory Care
Clinical Practice Guidelines

- **Blood Gas Analysis and Hemoximetry: 2001 Revision & Update**
- **Body Plethysmography: 2001 Revision & Update**
- **Exercise Testing for Evaluation of Hypoxemia and/or Desaturation: 2001 Revision & Update**
- **Methacholine Challenge Testing: 2001 Revision & Update**
- **Static Lung Volume: 2001 Revision & Update**

Respir Care 2001;46(5):498-539

Previously Published Guidelines:

- Removal of the Endotracheal Tube
- Single-Breath Carbon Monoxide Diffusing Capacity, 1999 Update
- Suctioning of the Patient in the Home
- Selection of Device, Administration of Bronchodilator, and Evaluation of Response to Therapy in Mechanically Ventilated Patients

Respir Care 1999;44(1):85-113

- Spirometry, 1996 Update
- Selection of an Oxygen Delivery Device for Neonatal and Pediatric Patients
- Selection of a Device for Delivery of Aerosol to the Lung Parenchyma
- Training the Health-Care Professional for the Role of Patient and Caregiver Educator
- Providing Patient and Caregiver Training

Respir Care 1996;41(7):629-663

- Assessing Response to Bronchodilator Therapy at Point of Care
- Discharge Planning for the Respiratory Care Patient
- Long-Term Invasive Mechanical Ventilation in the Home
- Capnography/Capnometry during Mechanical Ventilation
- Selection of an Aerosol Delivery Device for Neonatal and Pediatric Patients
- Polysomnography

Respir Care 1995;40(12):1300-1343

- Defibrillation during Resuscitation
- Management of Airway Emergencies
- Infant/Toddler Pulmonary Function Tests

Respir Care 1995;40(7):744-768

- Metabolic Measurement Using Indirect Calorimetry during Mechanical Ventilation
- Transcutaneous Blood Gas Monitoring for Neonatal and Pediatric Patients
- Capillary Blood Gas Sampling for Neonatal and Pediatric Patients
- Body Plethysmography

Respir Care 1994;39(12):1170-1190

- Ventilator Circuit Changes
- Delivery of Aerosols to the Upper Airway
- Neonatal Time-Triggered, Pressure-Limited, Time Cycled Mechanical Ventilation
- Application of Continuous Positive Airway Pressure to Neonates via Nasal Prongs or Nasopharyngeal Tube
- Surfactant Replacement Therapy
- Static Lung Volumes

Respir Care 1994;39(8):797-836

- Transport of the Mechanically Ventilated Patient
- Fiberoptic Bronchoscopy Assisting
- Resuscitation in Acute Care Hospitals
- Intermittent Positive Pressure Breathing
- Bland Aerosol Administration

Respir Care 1993;38(11):1169-1200

- Directed Cough
- Endotracheal Suctioning of Mechanically Ventilated Adults and Children with Artificial Airways
- In-Vitro pH and Blood Gas Analysis and Hemoximetry
- Single-Breath Carbon Monoxide Diffusing Capacity
- Use of Positive Airway Pressure Adjuncts to Bronchial Hygiene Therapy

Respir Care 1993;38(5):495-521

- Patient-Ventilator System Checks
- Humidification during Mechanical Ventilation
- Selection of Aerosol Delivery Device
- Nasotracheal Suctioning
- Bronchial Provocation
- Exercise Testing for Evaluation of Hypoxemia and/or Desaturation
- Arterial Blood Gas Sampling
- Oxygen Therapy in the Home or Extended Care Facility

Respir Care 1992;37(8):882-922

- Incentive Spirometry
- Pulse Oximetry
- Oxygen Therapy in the Acute Care Hospital
- Spirometry
- Postural Drainage Therapy

Respir Care 1991;36(12):1398-1426

AARC Clinical Practice Guideline

Blood Gas Analysis and Hemoximetry: 2001 Revision & Update

BGA 1.0 PROCEDURE:
Blood gas and pH analysis and hemoximetry (ie, CO-oximetry)

BGA 2.0 DESCRIPTION:
Analysis of arterial and/or mixed venous blood provides information concerning the oxygenation, ventilatory, and acid-base status of the subject from whom the specimen was obtained. Analysis of samples from other sources (ie, capillary, peripheral venous, umbilical venous samples, and pH measured from other body fluids) may provide limited information The variables most generally measured are the partial pressures for carbon dioxide and oxygen (P_{CO_2} and P_{O_2}), and hydrogen ion concentration (pH). Additional clinically useful variables are the concentration of total hemoglobin (tHb), oxyhemoglobin saturation (O_2Hb), and saturations of the dyshemoglobins (carboxyhemoglobin, or COHb, and methemoglobin, or metHb),[1-5] and other calculated or derived values such as plasma bicarbonate and base excess/deficit.

BGA 3.0 SETTING:
Analysis should be performed by trained individuals[4,6] in a variety of settings including, but not limited to:
3.1 hospital laboratories,
3.2 hospital emergency areas,
3.3 patient-care areas,
3.4 clinic laboratories,
3.5 laboratories in physicians' offices.[7]

BGA 4.0 INDICATIONS:
Indications for blood gas and pH analysis and hemoximetry include:
    4.1 the need to evaluate the adequacy of a patient's ventilatory (P_{aCO_2}), acid-base (pH and P_{aCO_2}), and/or oxygenation (P_{aO_2} and O_2Hb) status, the oxygen-carrying capacity (P_{aO_2}, O_2Hb, tHb, and dyshemoglobin saturations)[1,2,4] and intrapulmonary shunt (Q_{sp}/Q_t);
4.2 the need to quantitate the response to therapeutic intervention (eg, supplemental oxygen administration, mechanical ventilation) and/or diagnostic evaluation (eg, exercise desaturation);[1-3]
4.3 the need to monitor severity and progression of documented disease processes.[1,2]

BGA 5.0 CONTRAINDICATIONS:
Contraindications to performing pH-blood gas analysis and hemoximetry include:
    5.1 an improperly functioning analyzer;
5.2 an analyzer that has not had functional status validated by analysis of commercially prepared quality control products or tonometered whole blood[5,8-10] or has not been validated through participation in a proficiency testing program(s);[5,8,10-13]
5.3 a specimen that has not been properly anticoagulated;[4,5,14]
5.4 a specimen containing visible air bubbles;[1,4]
5.5 a specimen stored in a plastic syringe at room temperature for longer than 30 minutes, stored at room temperature for longer than 5 minutes for a shunt study, or stored at room temperature in the presence of an elevated leukocyte or platelet count (P_{aO_2} in samples drawn from subjects with very high leukocyte counts can decrease rapidly. Immediate chilling and analysis is necessary).[4,15-20]
5.6 an incomplete requisition that precludes adequate interpretation and documentation of results and for which attempts to obtain additional information have been unsuccessful. Requisitions should contain
    5.6.1 patient's name or other unique identifier, such as medical record number; birth date or age, date and time of sampling;

5.6.2 location of patient;

5.6.3 name of requesting physician or authorized individual;

5.6.4 clinical indication and tests to be performed;

5.6.5 sample source (arterial line, central venous catheter, peripheral artery);

5.6.6 respiratory rate and for the patient on supplemental oxygen fractional concentration of inspired oxygen (F_{IO_2}) or oxygen flow;

5.6.7 ventilator settings for mechanically ventilated patients (tidal volume, respiratory rate, F_{IO_2}, mode);

5.6.8 signature of person who obtained sample.[4,6]

It may also be useful to note body temperature, activity level, and working diagnosis. Test requisition should be electronically generated or handwritten and must be signed by the person ordering the test. Oral requests must be supported by written authorization within 30 days.[6]

5.7 an inadequately labeled specimen lacking the patient's full name or other unique identifier (eg, medical record number), date, and time of sampling.[4,5]

BGA 6.0 HAZARDS/COMPLICATIONS:

Possible hazards or complications include:

6.1 infection of specimen handler from blood carrying the human immunodeficiency virus, or HIV, hepatitis B, other blood-borne pathogens;[5,7,9,21]

6.2 inappropriate patient medical treatment based on improperly analyzed blood specimen or from analysis of an unacceptable specimen or from incorrect reporting of results.

BGA 7.0 LIMITATIONS OF PROCEDURE/ VALIDATION OF RESULTS:

7.1 Limitations of technique or methodology can limit value of the procedure. Erroneous results can arise from

7.1.1 sample clotting due to improper anticoagulation or improper mixing;[1,5,22]

7.1.2 sample contamination by

7.1.2.1 air,

7.1.2.2 improper anticoagulant and/or improper anticoagulant concentration,

7.1.2.3 saline or other fluids (specimen obtained via an indwelling catheter),

7.1.2.4 inadvertent sampling of systemic venous blood;

7.1.3 deterioration or distortion of variables to be measured resulting from

7.1.3.1 delay in sample analysis (Section 5.5);

7.1.3.2 inappropriate collection and handling (Accurate total hemoglobin concentration measurement depends on homogeneous mixture of specimen, appropriate anticoagulant concentration and specimen-size ratio, and absence of contamination of specimen by analyzer solutions or calibration gases. The concentration measured may also be dependent on the method incorporated by the specific analyzer.[5]);

7.1.3.3 incomplete clearance of analyzer calibration gases and previous waste or flushing solution(s);[5]

7.1.4 Hyperlipidemia causes problems with analyzer membranes and may affect CO-oximetry.[6]

7.1.5 Appropriate sample size is determined by the type of anticoagulant[1,2,5] and the sample requirements of the analyzer(s).[9] Attempts should be made to keep sample sizes as small as is technically feasible to limit blood loss, particularly in neonates.[4]

7.1.6 Some calculated values may be in error (eg, calculated S_{aO_2} may not reflect O_2Hb in the presence of COHb and/or metHb and with changes in 2,3 DPG concentration).

7.1.7 Arterialized capillary samples may be adequate to assess acid-base disorders but may not adequately reflect patient oxygenation.

7.1.8 The laboratory must have a defined procedure for temperature correction of the measured results. Errors in the measurement of the patient's temperature may cause erroneous temperature-corrected results. If temperature-adjusted results are reported, the report should be clearly labeled as such, and the measured results at 37 °C must also be reported.[5]

7.2 Results of analysis can be considered valid if

7.2.1 analytic procedure conforms to rec-

ommended, established guidelines[5,9] and follows manufacturer's recommendations;

7.2.2 results of pH-blood gas analysis fall within the calibration range of the analyzer(s) and quality control product ranges.[6] If a result outside of the usual calibration range is obtained (eg. P_{aO_2} measured as 250 torr, but analyzer calibrated to 140 torr), the analyzer should be recalibrated to accommodate this unusual value (using "calibration override" function and high- or 100%-oxygen standard gas).

7.2.3 laboratory procedures and personnel are in compliance with quality control and recognized proficiency testing programs.[5,6,8,9]

7.3 If questionable results are obtained and are consistent with specimen contamination:

7.3.1 the labeling of the blood sample container should be rechecked for patient's full name, medical record number (patient identifier), date and time of acquisition, and measured F_{IO_2} (or supplemental oxygen liter flow);[4,5]

7.3.2 the residual specimen should be reanalyzed (preferably on a separate analyzer);

7.3.3 an additional sample should be ob-

tained if the discrepancy cannot be resolved;

7.3.4 results of analysis of discarded samples should be logged with reason for discarding.[6]

BGA 8.0 ASSESSMENT OF NEED:

The presence of a valid indication (BGA 4.0) in the subject to be tested supports the need for sampling and analysis.

BGA 9.0 ASSESSMENT OF QUALITY OF TEST AND VALIDITY OF RESULTS:

The consensus of the committee is that all diagnostic procedures should follow the quality model described in the NCCLS GP26 A Quality System Model for Health Care.[23] (Fig. 1) The document describes a laboratory path of workflow model that incorporates all the steps of the procedure. This process begins with patient assessment and the generation of a clinical indication for testing through the application of the test results to patient care. The quality system essentials defined for all healthcare services provide the framework for managing the path of workflow. A continuation of this model for respiratory care services is further described in NCCLS HS4-A A Quality System Model for Respiratory
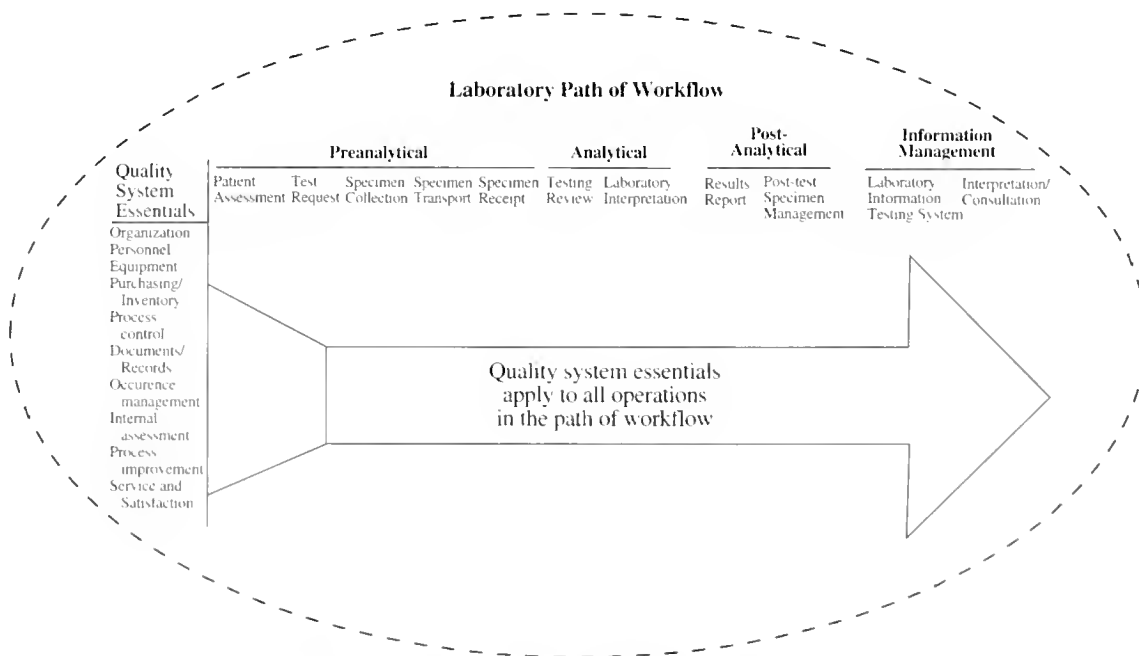

Fig. 1. Structure for a Quality System Model for a Laboratory Service (From Reference 23, with permission)

Care.[24] In both quality models the patient is the central focus.

9.1 General consideration include:

9.1.1 As part of any quality assurance program, indicators must be developed to monitor areas addressed in the path of workflow.

9.1.2 Each laboratory should standardize procedures and demonstrate intertechnologist reliability. Test results can be considered valid only if they are derived according to and conform to established laboratory quality control, quality assurance, and monitoring protocols.

9.1.3 Documentation of results, therapeutic intervention (or lack of) and/or clinical decisions based on testing should be placed in the patient's medical record.

9.1.4 The mode of ventilation, the oxygen concentration, and the oxygen delivery device and the results of the pretest assessment should be documented.

9.1.5 Report of test results should contain a statement by the technician performing the test regarding test quality (including patient understanding of directions and effort expended) and, if appropriate, which recommendations were not met.

9.1.6 Test results should be interpreted by a physician, taking into consideration the clinical question to be answered.

9.1.7 Personnel who do not meet annual competency requirements or whose competency is deemed unacceptable as documented in an occurrence report should not be allowed to participate, until they have received remedial instruction and have been re-evaluated.

9.1.8 There must be evidence of active review of quality control, proficiency testing, and physician alert, or 'panic' values, on a level commensurate with the number of tests performed

9.2 Blood gas-pH analysis and hemoximetry are beneficial only if no preanalytical error has occurred.[5]

9.3 Considerations related to equipment quality control and control materials:

9.3.1 For internal-equipment quality control using commercial controls:

9.3.1.2 Establish the mean and standard deviation (SD) for each constituent (ie, pH, P_{CO_2}, P_{O_2}) in each level for a new lot number of commercial quality control material prior to expiration of the old lot number. The laboratory director or designee should determine the acceptable range for quality control results based on statistically relevant or medical-needs criteria.

9.3.1.3 The frequency of each control run and number of levels is dependent on regulatory requirements and manufacturer's recommendations beyond a minimum of 1 level every 8 hours and 2 levels each day that the instrument is in operation.[6]

9.3.1.4 Quality control results outside predefined acceptability limits should trigger equipment troubleshooting. Quality control must be verified to be "in control" prior to analysis of specimens. Appropriate documentation of actions taken and results of verification are required.

9.3.1.5 Duplicate specimen analysis (ie, twice on one instrument or once on two instruments) may also be performed on a regular basis as an additional method of quality control. Duplicate analysis of the same analytes on different models of equipment is generally required by accrediting agencies.

9.3.1.6 Tonometry is the reference procedure to establish accuracy for blood P_{O_2} and P_{CO_2}. If issues of true accuracy arise, tonometry should be available.[5,25]

9.3.1.7 Electronic quality control monitors only the equipment performance. The use of nonelectronic controls at periodic intervals should also be employed to evaluate the testing process.[5]

9.3.1.8 Record keeping. Summarize all quality control data for a specified lot number. Maintain and generate reports according to regulatory and institutional policy.

9.3.2 External quality control or proficiency testing[5] considerations:

9.3.2.1 Proficiency testing is required by the Clinical Laboratory Amendments of 1988 (CLIA'88)[6] for each regulated analyte. Specimens of unknown values from an external source are to be analyzed a minimum of 3 times a year.

9.3.2.2 Proficiency-testing materials should be obtained from an approved source to meet regulatory requirements.

9.3.2.3 The proficiency testing survey report should be carefully reviewed by the medical director and laboratory supervisor. If the results are suboptimal, the medical director and supervisor should promptly review their equipment, procedures, and materials to ascertain the cause of the poor performance.[24]

9.3.3 With new equipment installation:[24]

9.3.3.1 CLIA '88 requires the evaluation of equipment accuracy and imprecision prior to analysis of patient samples.[6]

9.3.3.2 Tonometry is the reference method for establishing accuracy for P_{aO_2} and P_{aCO_2},[25] but unless the entire tonometry process is of the highest quality, it, too, can have errors.

9.3.3.3 When an existing instrument is replaced, duplicate analysis must be performed to compare the new instrument to the existing instrument.

9.3.4 Calibration verification[24]

9.3.4.1 Calibration verification is performed prior to initial use and at 6-month intervals. Calibration verification is completed by analyzing a minimum of 3 levels of control material to verify the measuring range of the analyzer. A fourth level should be considered if samples with high O_2 levels are analyzed on the instrument.

9.3.4.2 Frequency of calibration verification may vary according to regulatory agencies under which the laboratory is accredited or licensed [ie, College of American Pathologists (CAP), CLIA'88 or Joint Commission on Ac-

creditation of Healthcare Organizations (JCAHO)].

9.4 Testing (analytical phase) is carried out according to an established proven protocol, conforming to manufacturer recommendations;[5,9] The following aspects of analysis should be monitored and corrective action taken as indicated:

9.4.1 detection of presence of air bubbles or clots in specimen, with evacuation prior to mixing and sealing of syringe;[1,4,5]

9.4.2 assurance that an uninterrupted (ie, solid or continuous) sample is aspirated (or injected) into analyzer and that all of the electrodes are covered by the sample (confirmed by direct viewing of sample chamber if possible;[9]

9.4.3 assurance that 8-hour quality control and calibration procedures have been completed and that instrumentation is functioning properly prior to patient sample analysis;[5,6,8]

9.4.4 assurance that specimen was properly labeled, stored, and analyzed within an acceptable period of time[4,5] (see Section 5.5).

9.5 Post-testing (post-analytical phase) The results should validate or contradict the patient's clinical condition (ie, the basis for ordering the test).[26,27]

9.5.1 Documentation of results, therapeutic intervention (or lack of), and/or clinical decisions based upon the pH-blood gas measurements should be available in the patient's medical record and/or be otherwise readily accessible (eg, at the testing area) for at least 2 years.[6]

9.5.2 Reference intervals and 'critical values' must be determined for each analyte prior to sample analysis. If the reference interval is determined by transference, the interval should be validated. Defining and determining reference intervals is described in NCCLS document C24-A2.[28]

BGA 10.0 RESOURCES:

Federal regulations,[6] stipulate that requirements relative to personnel (levels of education and training), documentation procedures and equipment be fulfilled. Blood gas instrumentation is classified as being either moderately or highly complex. Persons performing blood gas analysis should be

conversant with applicable federal regulations (CLIA'88)[6] and appropriately qualified.

10.1 Recommended Equipment:

10.1.1 Automated or semiautomated pH-blood gas analyzer with related calibration gases, electrodes, membranes, electrolytes, reagents, and accessories.[5,6,9]

10.1.2 Fixed, multiple wavelength spectrophotometer (hemoximeter or CO-oximeter)[7] or other device for determining total hemoglobin and its components.

10.1.3 Protective eye wear as necessary and outer wear, protective gloves, impenetrable needle container, face mask and/or face-shield.[29]

10.1.4 Quality control and proficiency testing materials.

10.2 Personnel:

The following recommendations are for tests of moderate complexity, as designated by CLIA '88.[6] Persons at either of the levels described should perform pH-blood gas analysis under the direction and responsibility of a laboratory director and technical consultant (may be the same individual) who possess at least a baccalaureate degree and who have specific training in blood gas analysis and interpretation.[6]

10.2.1 Level 1: Personnel should be specifically trained in pH-blood gas analysis, oxygen delivery devices, and related equipment, record keeping, and hazards and sources of specimen and handler contamination(s) associated with sampling and analysis. Such persons should be, at minimum, high school graduates (or equivalent) with strong backgrounds in mathematics, and preferably with one or more years of college courses in the physical and biological sciences.[30] Such persons must have documented training and demonstrated proficiency in pH-blood gas analysis, preventive maintenance, troubleshooting, instrument calibration, and awareness of the factors that influence test results, and the skills required to verify the validity of test results through the evaluation of quality-control sample values, prior to analyzing patient specimens and reporting results[6,30] Performance of

pH-blood gas analysis must be supervised by a Level-II technologist.

10.2.2 Level II: Level-II personnel supervise Level-I personnel and are health care professionals specifically trained (with proven, documented proficiency) in all aspects of blood gas analysis and hemoximetry:

10.2.2.1 quality control, quality assurance, and proficiency testing;

10.2.2.2 operation and limitations, including instrument troubleshooting and appropriate corrective measures.

10.2.2.3 Level-II personnel should be cognizant of various means for specimen collection and the causes and impact of preanalytical and instrument error(s).

10.2.2.4 Level-II personnel should be trained in patient assessment, acid-base and oxygenation disorders, and diagnostic and therapeutic alternatives. A baccalaureate, or higher, degree in the sciences or substantial experience in pulmonary function technology is preferred. Although, 2 years of college in biological sciences and mathematics, plus 2 years of training and experience, or equivalent may be substituted for personnel supervising arterial pH-blood gas analysis.[30] A recognized credential (MT, MLT, CRT, RRT, CPFT, RPFT) is strongly recommended.[6]

BGA 11.0 MONITORING:

Monitoring of personnel, sample handling, and analyzer performance to assure proper handling, analysis, and reporting should be ongoing, during the process.

BGA 12.0 FREQUENCY:

Frequency of execution of procedures depends upon the sample load of the laboratory and the requirements of agencies that specify quality control maneuvers.

BGA 13.0 INFECTION CONTROL:

13.1 The staff, supervisors, and physician-directors associated with the pulmonary laborato-

ry should be conversant with "Guideline for Isolation Precautions in Hospitals" made by the Centers for Disease Control and the Hospital Infection Control Practices Advisory Committee (HICPAC).[31] and develop and implement policies and procedures for the laboratory that comply with its recommendations for Standard Precautions and Transmission-Based Precautions.

13.2. The laboratory's manager and its medical director should maintain communication and cooperation with the institution's infection control service and the personnel health service to help assure consistency and thoroughness in complying with the institution's policies related to immunizations, post-exposure prophylaxis, and job- and community-related illnesses and exposures.[32]

13.3 Primary considerations include

13.3.1 adequate handwashing, [33]

13.3.2 provision of prescribed ventilation with adequate air exchanges.[34]

13.3.3 careful handling and thorough cleaning and processing of equipment.[31]

13.3.4 the exercise of particular care in scheduling and interfacing with the patient in whom a diagnosis has not been established.[31,34]

BGA 14.0 AGE-SPECIFIC ISSUES:
This document applies to samples from neonatal, pediatric, adult, and geriatric populations.

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REFERENCES

1. Shapiro BA, Peruzzi WT, Kozelowski-Templin R. Clinical application of blood gases, 5th ed. St Louis: Mosby-Year Book Inc; 1994.

2. Raffin TA. Indications for arterial blood gas analysis. Ann Intern Med 1986;105(3):390-398.

3. Browning JA, Kaiser DL, Durbin CG Jr. The effect of guidelines on the appropriate use of arterial blood gas analysis in the intensive care unit. Respir Care 1989;34(4):269-276.

4. National Committee for Clinical Laboratory Standards (NCCLS). Procedures for the collection of arterial blood specimens, 1999. Available from NCCLS: phone 610-688-0100; Fax 610-688-0700; e-mail exoffice@nccls.org.

5. National Committee for Clinical Laboratory Standards (NCCLS) C46-P Blood gas and pH analysis and related measurements: proposed guideline, 2000. Available, when approved, from NCCLS: phone 610-688-0100; Fax 610-688-0700; e-mail exoffice@nccls.org.

6. Department of Health & Human Services Health Care Financing Administration Public Health Service 42 CFR Part 405, et al. Clinical laboratory improvement amendments of 1988; final rule. Federal Register Friday February 28, 1992.

7. Task Force on Guidelines, Society of Critical Care Medicine. Recommendations for services and personnel for delivery of care in a critical care setting. Crit Care Med 1988;16(8):809-811.

8. Moran RF. Assessment of quality control of blood gas/pH analyzer performance. Respir Care 1981;26(6):538-546.

9. Assessing respiratory status with blood gas/pH analyzers and in vitro multiwavelength oximeters. Health Devices 1989; 18(7-8):239-284.

10. Elser RC. Quality control of blood gas analysis: a review. Respir Care 1986;31(9):807-816.

11. Ehrmeyer S, Laessig RH. Measurement of the proficiency of pH and blood gas analyses by interlaboratory proficiency testing. J Med Tech 1985;2:33-38.

12. Hansen JE, Jensen RL, Casaburi R, Crapo RO. Comparison of blood gas analyzer biases in measuring tonometered blood and a fluorocarbon-containing, proficiency-testing material. Am Rev Respir Dis 1989;140(2):403-409.

13. Hansen JE, Casaburi R, Crapo RO, Jensen RL. Assessing precision and accuracy in blood gas proficiency testing. Am Rev Respir Dis 1990;141(5 Pt 1):1190-1193.

14. Cissik JH, Salustro J, Patton OL, Louden JA. The effects of sodium heparin on arterial blood-gas analysis. CVP Jan/Feb 1977.

15. Muller-Plathe O, Heyduck S. Stability of blood gases, electrolytes and hemoglobin in heparinized whole blood samples: influence of the type of syringe. Eur J Clin Chem Clin Biochem 1992;30(6):349-355.

16. Wu FY, Barazanji KW, Johnson RL. Source of error on A-aDO2 calculated from blood stored in plastic and glass syringes. J Appl Physiol 1997;82(1):196-202.

17. Mahoney JJ, Harvey JA, Wong RJ, Van Kessel AI. Changes in oxygen measurements when whole blood is stored in iced plastic or glass syringes. Clin Chem 1991;37(7):1244-1248.

18. Smeenk FW, Janssen JD, Arends BJ, Harff GA, van den Bosch JA, Schonberger JP, Postmus PE. Effects of four different methods of sampling arterial blood and storage time on gas tensions and shunt calculation in the 100% oxygen test. Eur Respir J 1997;10(4):910-913.

19. Hooton TM. Protecting ourselves and our patients from nosocomial infections. Respir Care 1989;34(2):111-115.

20. Koepke JA. Additives to blood collection: heparin. National Committee for Clinical Laboratory Standards 1988;H24-T Vol. 8;No. 5 33-55.

21. Fox MJ, Brody JS, Weintraub LR. Leukocyte larceny: a cause of spurious hypoxemia. Am J Med 1979;67(5):742-746.

22. Matchuny JK. Unexplained hypoxemia in a leukemia patient (Blood Gas Corner). Respir Care 1988;33(10):971-973.

23. National Committee for Clinical Laboratory Standards (NCCLS). GP26-A A quality system model for health care: approved guideline (1999). Available from NCCLS: phone 610-688-0100; Fax 610-688-0700; e-mail exoffice@ncels.org.

24. National Committee for Clinical Laboratory Standards (NCCLS). NCCLS HS4-A A quality system model for respiratory care. Available from NCCLS: phone 610-688-0100; Fax 610-688-0700; e-mail exoffice@ncels.org.

25. Burnett RW, Covington AK, Maas AHJ, et al. IFCC method for tonometry of blood: reference materials for PCO2 and PO2. An approved IFCC recommendation. J Biomed Lab Sci 1989;2:185-192.

26. American Thoracic Society. ATS pulmonary function laboratory management and procedure manual. New York: ATS, 1998.

27. Thorson SH, Marini JJ, Pierson DJ, Hudson LD. Variability of arterial blood gas values in stable patients in the ICU. Chest 1983;84(1):14-18.

28. National Committee for Clinical Laboratory Standards (NCCLS). C24-A2.

29. Occupational Safety & Health Administration. Occupational exposure to bloodborne pathogens. Final Rule 29 CFR Part 1910.1030. Federal Register Friday, December 06, 1991.

30. Gardner RM, Clausen JL, Epler G, Hankinson JL, Permutt S, Plummer AL. Pulmonary function laboratory personnel qualifications. American Thoracic Society Position Paper. ATS News; November 1982.

31. Garner JS. Hospital Infection Control Practices Advisory Committee, Centers for Disease Control and Prevention. Guideline for isolation precautions in hospitals. Am J Infect Control 1996;24(1):24-31. or http//www.apic.org/html/rese/gdisolat.html.

32. Centers for Disease Control and Prevention, Hospital Infection Control Practices Advisory Committee. Guideline for infection control in health care personnel, 1998. Am J Infect Control 1998;26:269-354 or Infect Control Hosp Epidemiol 1998;19(6):407-463.

33. Larson EL. APIC guideline for handwashing and hand antisepsis in health care settings. Am J Infect Control 1995;23(4):251-269.

34. Centers for Disease Control & Prevention. Guidelines for preventing the transmission of Mycobacterium tuberculosis in health-care facilities, 1994. MMWR 1994;43(RR13):1-32 or Federal Register 1994;59(208):54242-54303 or http://aepo-xdv-www.epo.cdc.gov/wonder/prevguid/m0035909/m0035909.htm

AARC Clinical Practice Guideline

Body Plethysmography: 2001 Revision & Update

BP 1.0 PROCEDURE:
Body plethysmography for determination of thoracic gas volume (VTG) and airways resistance (R_{aw}).

BP 2.0 DESCRIPTION/DEFINITION:
During body plethysmography, the subject is enclosed in a chamber equipped to measure pressure, flow, or volume changes. The most common measurements made using the body plethysmograph are VTG and R_{aw}.[1,2] Airways conductance (G_{aw}) is also commonly calculated as the reciprocal of R_{aw}. Specific airways conductance (ie, conductance/unit of lung volume) is routinely reported as sG_{aw}. Other tests that can be administered in the body plethysmograph include spirometry, bronchial challenge, diffusing capacity (D_{LCO}), single-breath nitrogen (N_2), multiple-breath N_2 washout, pulmonary compliance, and occlusion pressure. These will not be discussed as part of this guideline. Some have been previously addressed.[3-6]

> 2.1 VTG is expressed in liters (BTPS, or body temperature and pressure saturated) and is the volume of gas in the lung when the mouth shutter is closed. In plethysmographic studies, it is commonly used to represent the functional residual capacity (FRC).
>
> 2.2 R_{aw} is reported in cm H_2O/L/s (ie, cm $H_2O \cdot L^{-1} \cdot s^{-1}$).
>
> 2.3 sG_{aw} is reported in L/s/cm H_2O (ie, $L \cdot s^{-1} \cdot$ cm H_2O^{-1}) and is the reciprocal of the R_{aw} ($1/R_{aw}$) divided by the lung volume at which the resistance measurement is made.

BP 3.0 SETTINGS:
> 3.1 Pulmonary function laboratories
> 3.2 Cardiopulmonary laboratories
> 3.3 Clinics and physician's offices

BP 4.0 INDICATIONS:
Body plethysmographic determination of VTG, R_{aw}, and sG_{aw} may be indicated:

> 4.1 for diagnosis of restrictive lung disease;
>
> 4.2 for measurement of lung volumes to distinguish between restrictive and obstructive processes;
>
> 4.3 for evaluation of obstructive lung diseases, such as bullous emphysema and cystic fibrosis, which may produce artifactually low results if measured by helium dilution or N_2 washout.[7] With simultaneously determined volumes, an index of trapped gas (ie, $FRC_{plethysmograph}/FRC_{He\ dilution}$) can be established.[8]
>
> 4.4 for measurement of lung volumes when multiple repeated trials are required or when the subject is unable to perform multibreath tests;[9]
>
> 4.5 for evaluation of resistance to airflow;[10]
>
> 4.6 for determination of the response to bronchodilators, as reflected by changes in R_{aw}, sG_{aw}, and VTG;[11]
>
> 4.7 for determination of bronchial hyperreactivity in response to methacholine, histamine, or isocapnic hyperventilation as reflected by changes in VTG, R_{aw}, and sG_{aw};[12,13]
>
> 4.8 for following the course of disease and response to treatment.

BP 5.0 CONTRAINDICATIONS:
Relative contraindications to body plethysmography are:

> 5.1 mental confusion, muscular incoordination, body casts, or other conditions that prevent the subject from entering the plethysmograph cabinet or adequately performing the required maneuvers (ie, panting against a closed shutter);
>
> 5.2 claustrophobia that may be aggravated by entering the plethysmograph cabinet;
>
> 5.3 presence of devices or other conditions, such as continuous intravenous infusions with pumps or other equipment that will not fit into the plethysmograph, that should not be discontinued, or that might interfere with pressure

changes (eg, chest tube, transtracheal O_2 catheter, or ruptured eardrum);

5.4 continuous oxygen therapy that should not be temporarily discontinued.

BP 6.0 HAZARDS/COMPLICATIONS:

6.1 VTG and R_{aw} measurements require the subject to pant against a closed shutter; improper panting technique may result in excessive intrathoracic pressures.

6.2 Prolonged confinement in the plethysmograph chamber could result in hypercapnia or hypoxia; however, because of the limited length of the test and the fact that the plethysmograph must be vented periodically, this is an uncommon occurrence.

6.3 Transmission of infection is possible via improperly cleaned equipment (ie, mouthpieces) or as a consequence of the inadvertent spread of droplet nuclei or body fluids (patient-to-patient or patient-to-technologist).

BP 7.0 LIMITATIONS OF METHODOLOGY/ VALIDATION OF RESULTS:

Limitations of the body plethysmograph in measurement of VTG, R_{aw}, and sG_{aw} include but are not limited to:

7.1 overestimation of VTG in subjects with severe obstruction or induced bronchospasm unless a slow 'panting' speed (ie, approximately 1 cycle/s) is maintained.[14-17]

7.2 Erroneous measurement of VTG, R_{aw}, or sG_{aw} due to improper panting technique. Excessive pressure fluctuations or signal drift during panting may invalidate VTG, R_{aw}, or sG_{aw}.[18]

7.3 Nonpanting measurements have been suggested for use in children or others who have difficulty mastering the panting maneuver.[19,20] Nonpanting maneuvers in plethysmographs with built-in thermal leaks may invalidate VTG or R_{aw} measurements.[2,21]

7.4 Computer-determined slopes of either VTG or R_{aw} tangents may be inaccurate. Many systems calculate the slopes using a best-fit regression analysis. This technique may produce widely varying results if extraneous data points are included (due to improper panting or excessive signal drift). All slopes should be visually inspected and adjusted according to an estab-

lished laboratory procedure.

7.5 Excessive abdominal gas or panting techniques that employ accessory muscles may increase the measured VTG, due to compression effects.[24]

7.6 Plethysmography is a complex test. Careful calibration of multiple transducers is required. Attention to frequency response, thermal stability, and leaks is necessary.[25]

7.7 Choice and application of reference values affect interpretation. Reference values for VTG using plethysmographically determined lung volumes are not widely available.

> **7.7.1** Make a tentative selection from whatever published reference values are available. The characteristics of the healthy reference population should match the study group with respect to age, body size, gender, and race. The equipment, techniques, and measurement conditions should be similar.
>
> **7.7.2** Following selection of seemingly appropriate reference values, compare measurements obtained from a representative sample of healthy individuals (10-20 subjects, over an appropriate age range) to the predicted values obtained from the selected reference values. If an appreciable number of the sample fall outside of the normal range, more appropriate reference values should be sought. This procedure detects only relatively gross differences between sample and reference populations.[26]

BP 8.0 ASSESSMENT OF NEED:

8.1 See Section 4.0 Indications.

8.2 Protocols may define the need for measurement of lung volumes and airway resistance measurements based on the results of previously performed tests (ie, spirometry, diffusing capacity) and the clinical question to be answered.

BP 9.0 ASSESSMENT OF QUALITY & VALIDATION OF RESULTS:

The consensus of the Committee is that all diagnostic procedures should follow the quality model described in the NCCLS GP26-A A Quality System Model for Health Care.[27] (Fig. 1) The document de-

scribes a laboratory path of workflow model that incorporates all the steps of the procedure. This process begins with patient assessment and the generation of a clinical indication for testing through the application of the test results to patient care. The quality system essentials defined for all health care services provide the framework for managing the path of workflow. A continuation of this model for respiratory care services is further described in NCCLS HS4-A A Quality System Model for Respiratory Care.[28] In both quality models the patient is the central focus.

9.1.3 Documentation of results, therapeutic intervention (or lack of) and/or clinical decisions based on the testing should be placed in the patient's medical record.

9.1.4 The type of medications, dose, and time taken prior to testing and the results of the pretest assessment should be documented.

9.1.5 Report of test results should contain a statement by the technician performing the test regarding test quality (including patient understanding of directions and ef-

Pulmonary Diagnostics Path of Workflow

Fig. 1. Structure for a Quality System Model for a Pulmonary Diagnostics Service (From Reference 27, with permission)

9.1 General consideration include:

    9.1.1 As part of any quality assurance program, indicators must be developed to monitor areas addressed in the path of workflow.

    9.1.2 Each laboratory should standardize procedures and demonstrate intertechnologist reliability. Test results can be considered valid only if they are derived according to and conform to established laboratory quality control, quality assurance, and monitoring protocols.

fort expended) and, if appropriate, which recommendations were not met.[29-31]

9.1.6 Test results should be interpreted by a physician, taking into consideration the clinical question to be answered.

9.1.7 Personnel who do not meet annual competency requirements or whose competency is deemed unacceptable as documented in an occurrence report should not be allowed to participate, until they have received remedial instruction and have

been re-evaluated.

9.1.8 There must be evidence of active review of quality control, proficiency testing, and physician alert, or 'panic' values, on a level commensurate with the number of tests performed.

9.2 Calibration and quality control measures specific to equipment used in plethysmography include:

9.2.1 Calibration at recommended frequencies, at any time accuracy is suspect, and when the equipment is moved to a different location.

9.2.2 On a daily basis, calibrate volume, mouth and box pressure.

9.2.3 At least monthly, manually calibrate systems in addition to daily use of the autocalibration system.

9.2.4 At least weekly, assess linearity of flow-sensing device.

9.2.5 At least quarterly, perform airway resistance with a known resistor and calculate results.[32]

9.2.6 At least annually or at a frequency established by the laboratory on the basis of the tendency of the device to vary, check volume with isothermal bottle.[33]

9.2.7 At least monthly and at any time accuracy is suspect, perform tests on standard subjects (biologic controls, or bio-QC).[29,32]

9.2.8 Test standard subjects more frequently initially to establish statistical variation for comparison.

9.2.9 It may be advantageous to perform Bio-QC at weekly or semi-monthly intervals.

9.3 Test Quality Assessment: Results are valid if the equipment functions correctly and the subject is able to perform acceptable and reproducible maneuvers.

9.3.1 VTG maneuvers are acceptable when:

9.3.1.1 the displayed or recorded tracing indicates proper panting technique (the loop generated against a closed shutter should be closed or nearly so). The patient should support his/her cheeks with the hands to prevent pressure changes induced by the mouth.[34]

This should be done without supporting the elbows or elevating the shoulders.

9.3.1.2 Recorded pressure changes should be within the calibrated pressure range of each transducer (See Section 10.1.3). The entire tracing should be visible. Pressure changes that are too large or too small may yield erroneous results.

9.3.1.3 Thermal equilibrium should be evident; tracings should not drift on the display or recording. (This typically takes 1-2 minutes.)

9.3.1.4 The panting frequency is approximately 1 Hz. Nonpanting maneuvers may be acceptable if the plethysmograph system is specifically designed to perform such maneuvers.[14,35]

9.4. R_{aw} and sG_{aw} maneuvers may be considered acceptable if:

9.4.1 they meet criteria given in Sections 9.3.1.1 through 9.3.1.3;

9.4.2 the open-shutter panting maneuver shows a relatively closed loop, particularly in the range of +0.5 to -0.5 L/s;

9.4.3 the panting frequency during serial measurements in a given patient is kept constant to aid in interpretation. Consensus of the group suggests a range of 90-150 cycles per minute (1.5-2.5 Hz). Frequency should be held constant for within-testing session comparisons (ie, pre- and post-bronchodilator testing) and serial testing.

9.5 Test Results Reporting:

9.5.1 The reported VTG

9.5.1.1 should be averaged from a minimum of 3-5 separate, acceptable panting maneuvers;[36,37]

9.5.1.2 should be calculated using values that agree within 5% of the mean (widely varying values should be averaged, and reported as variable);

9.5.1.3 should indicate whether the thoracic volume was at FRC or at some other level;

9.5.1.4 should be compared with other lung volume determinations (He dilution, N_2 washout) if such are being performed;

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9.5.1.5 should be corrected for patient weight for some systems.

9.5.2 Lung Volumes including the slow vital capacity (VC) maneuver and its subdivisions inspiratory capacity (IC) and expiratory reserve volume (ERV) should be performed during the same testing session. The ERV, IC, and VC should be measured in conjunction with each VTG trial before disconnecting from the measuring system. Add tracing to illustrate correct performance.

> **9.5.2.1** The largest volume of VC or FVC obtained should be used for calculation of derived lung volumes (ie, total lung capacity, or TLC, residual volume, or RV, and RV/TLC%).
>
> **9.5.2.2** The mean values should be reported for IC and ERV from acceptable VTG maneuvers.
>
> **9.5.2.3** There are various methods to calculate TLC, but by consensus the Committee recommends use of:
> TLC = mean FRC + mean IC*
> *(Note: Mean IC should be close to the largest IC)
> RV = TLC – largest VC

9.6 The reported R_{aw} and sG_{aw}

> **9.6.1** should be calculated from the ratio of closed and open shutter tangents for each maneuver.[38] (Airway resistance and lung volume are interdependent in a nonlinear fashion):
>
> **9.6.2** should be averaged from 3-5 separate, acceptable maneuvers as calculated in 9.4; reproducibility should be based on sG_{aw} and the suggested limit for variance is within 10% of the mean; (eg, if the measured results are ≤ 0.17, accept ± 0.01 or if the measured results are ≥ 0.20, use ± 0.02)[39]
>
> **9.6.3** should have the open-shutter tangent (V/P_{box}) measured between flows of +0.5 and -0.5 L/s. For loops that display hysteresis, the inspiratory limbs may be used;[38]
>
> **9.6.4** should have the sG_{aw} calculated using the VTG at which the shutter was closed for each individual maneuver.[24]

9.7 Report of test results should contain a statement by the technologist performing the test concerning test quality and, if appropriate, which recommendations were not met.

9.8 Reference equations: Each laboratory should select reference equations appropriate for the methods and the population tested. Guidance for defining and determining reference intervals is provided in American Thoracic Society (ATS)[32] and NCCLS[40] documents.

9.9 Test quality monitoring: Plethysmography results should be subject to ongoing review by a supervisor, with feedback to the technologist. The monitoring should include visual inspection of the VTG and R_{aw} loops and fitted lines. Quality assurance (QA) and/or quality improvement (QI) programs should be designed to monitor the technologist both initially and on an ongoing basis.

BP 10.0 RESOURCES:

> **10.1** Equipment:
>
> > **10.1.1** Volume-measuring devices used in the plethysmograph (ie, the pneumotachometer) should meet or exceed ATS recommendations. A 3-L syringe should be available for calibration.[31]
> >
> > **10.1.2** Either pressure (constant volume) or flow-type plethysmographs may be used.
> >
> > **10.1.3** Transducers in the plethysmograph should meet prescribed range specifications:[24]
> > Mouth pressure: ±20 to 50 cm H_2O
> > Box pressure: ±2 cm H_2O (500-L box)
> > Flow: 0.2 to 1.5 L/s
> >
> > **10.1.4** Pressure and volumes signals should be phase aligned up to 10 Hz.
> >
> > **10.1.5** A plenum or similar device that facilitates thermal equilibrium is recommended. Some plethysmographs utilize air conditioning to maintain thermal equilibrium.
> >
> > **10.1.6** The plethysmograph cabinet should be easy for the subject to enter and exit. The door should preferably be operable from within the box. The cabinet should be equipped with an intercom and should provide adequate visibility for both the technologist and the subject.
> >
> > **10.1.7** The plethysmograph system, if computerized, should allow for technologist adjustment of open- and closed-shutter tangents.
> >
> > **10.1.8** Calibration devices should include (in addition to a 3-L syringe) 30-50 mL

sine-wave pump (variable speed, used primarily for calibration of pressure boxes), water manometer ±20 cm H_2O (used for calibration of the mouth pressure transducer), and rotameter 0 to 1.5 L/s (used for calibration of the pneumotachometer).

10.2 Personnel: Plethysmography should be performed under the direction of a physician trained in pulmonary function testing. It may be performed by technologists who meet criteria for either Level I or Level II. Plethysmographic results can be compromised if the test is performed by inadequately trained personnel.

> **10.2.1 Level I:** The technologist performing plethysmography should be a high school graduate or equivalent with a demonstrated ability to perform spirometry and lung volume determinations. Level I personnel should perform plethysmography only under the supervision of a Level II technologist or a physician.

> **10.2.2 Level II:** Personnel supervising plethysmography should have formal education and training.[41] This may be part of an accredited program in respiratory therapy or pulmonary function technology or 2 years of college work in biological sciences and mathematics. Level II personnel should also have 2 or more years experience performing spirometry, lung volumes, and diffusing capacity tests. Attainment of the credential of Certified Pulmonary Function Technologist (CPFT) or Registered Pulmonary Function Technologist (RPFT) is recommended.

BP. 11.0 PATIENT MONITORING:

(See also Section 9.0 Assessment of Quality)

11.1 Evaluate the patient's breathing pattern to verify a stable FRC level.

11.2 Verify appropriate shutter-closure timing.

11.3 Gauge the level of understanding (of test instructions), effort, and cooperation by the subject.

BP 12.0 FREQUENCY:

The frequency with which plethysmography is repeated should depend on the clinical question(s) to be answered.

BP 13.0 INFECTION CONTROL:

13.1 The staff, supervisors, and physician-directors associated with the pulmonary laboratory should be conversant with "Guideline for Isolation Precautions in Hospitals"[42] and develop and implement policies and procedures for the laboratory that comply with its recommendations for Standard Precautions and Transmission-Based Precautions.

13.2 The laboratory's manager and its medical director should maintain communication and cooperation with the institution's infection control service and the personnel health service to help assure consistency and thoroughness in complying with the institution's policies related to immunizations, post-exposure prophylaxis, and job- and community-related illnesses and exposures.[43]

13.3 Primary considerations include adequate handwashing,[44] provision of prescribed ventilation with adequate air exchanges,[45] careful handling and thorough cleaning and processing of equipment,[46] and the exercise of particular care in scheduling and interfacing with the patient in whom a diagnosis has not been established.[45] Considerations specific for plethysmography measurement include:

> **13.3.1** The use of filters is neither recommended nor discouraged. Filters may be appropriate for use in systems that use valves or manifolds on which deposition of expired aerosol nuclei is likely.[47]

> **13.3.2** If filters are used in gas-dilution procedures, their volume should be subtracted when FRC is calculated.

> **13.3.3** If filters are used in the plethysmograph system, the resistance of the filters should be subtracted from the airways resistance calculation.

> **13.3.4** Nondisposable mouthpieces and equipment parts that come into contact with mucous membranes, saliva, and expirate should be cleaned and sterilized or subjected to high-level disinfection between patients.[46] Gloves should be worn when handling potentially contaminated equipment.

> **13.3.5** Flow sensors, valves, and tubing not in direct contact with the patient should be routinely disinfected according to the hospital's infection control policy. Any equipment surface that displays visible condensation from expired gas should be disinfected or sterilized before it is reused.

> **13.3.6** Water-sealed spirometers should be drained weekly and allowed to dry.[30]

13.3.7 Closed circuit spirometers, such as those used for He-dilution FRC determinations, should be flushed at least 5 times over their entire volume to facilitate clearance of droplet nuclei. Open circuit system need only have the portion of the circuit through which rebreathing occurs decontaminated between patients.

14.0 AGE-SPECIFIC ISSUES:

Test instructions should be provided and techniques described in a manner that takes into consideration the learning ability and communications skills of the patient being served.

14.1 Neonatal: This Guideline does not apply to the neonatal population.

14.2 Pediatric: These procedures are appropriate for children who can perform spirometry of acceptable quality and can adequately follow directions for plethysmographic testing.

14.3. Geriatric: These procedures are appropriate for members of the geriatric population who can perform spirometry of acceptable quality and adequately follow directions for plethysmographic testing.

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REFERENCES

1. DuBois AB, Botelho SY, Bedell GN, Marshal R, Comroe JH. A rapid plethysmographic method for measuring thoracic gas volume: a comparison with nitrogen washout method for measuring functional residual capacity in normal subjects. J Clin Invest 1956;35:322.

2. DuBois AB, Botelho SY, Comroe JH. A new method for measuring airway resistance in man using a body plethysmograph: values in normal subjects and in patients with respiratory disease. J Clin Invest 1956;35:327.

3. American Association for Respiratory Care. Clinical Practice Guideline: Spirometry, 1996 update. Respir Care 1996;47(7):629-636.

4. American Association for Respiratory Care. Clinical Practice Guideline: Bronchial provocation: 2001 revision & update. Respir Care 2001;46(5):506-513.

5. American Association for Respiratory Care. Clinical Practice Guideline: Single-breath carbon monoxide diffusing capacity, 1999 update. Respir Care 1999;44(5):539-546.

6. American Association for Respiratory Care. Clinical Practice Guideline: Static lung volumes: 2001 revision & update. Respir Care 2001;46(5):531-539.

7. Ferris BG. Epidemiology Standardization Project (American Thoracic Society). Am Rev Respir Dis 1978;118(6, Pt 2):1-120.

8. Pearle JL. Correlation of helium and plethysmographic lung volumes in airways obstruction. Am Rev Diag 1983;2:47.

9. Miller WF, Scacci R, Gast LR. Laboratory evaluation of pulmonary function. Philadelphia: JB Lippincott; 1987.

10. Reinoso MA, Jett JR, Beck KC. Body plethysmography in the evaluation of intrathoracic airway abnormalities. Chest 1992;101(6):1674-1676.

11. Watanabe S, Renzetti AD Jr, Begin R, Bigler AH. Airway responsiveness to a bronchodilator aerosol. 1. Normal human subjects. Am Rev Respir Dis 1974;109(5):530-537.

12. Fish JE, Rosenthal RR, Batra G, Menkes H, Summer W, Permutt S, Norman P. Airway responses to methacholine in allergic and nonallergic subjects. Am Rev Respir Dis 1976;113(5):579-586.

13. Crapo RO, Casaburi R, Coates AL, Enright PL, Hankinson JL, Irvin CG, et al. Guidelines for methacholine and exercise challenge testing, 1999. This official statement of the American Thoracic Society was adopted by the ATS Board of Directors, July 1999. Am J Respir Crit Care Med 2000;161(1):309-329.

14. Rodenstein DO, Stanescu DC, Francis C. Demonstration of failure of body plethysmography in airway obstruction. J Appl Physiol 1982;52(4):949-954.

15. Shore SA, Huk O, Mannix S, Martin JG. Effect of panting frequency on the plethysmographic determination of thoracic gas volume in chronic obstructive pulmonary disease. Am Rev Respir Dis 1983;128(1):54-59.

16. Shore S, Milic-Emili J, Martin JG. Reassessment of body plethysmographic technique for the measurement of thoracic gas volume in asthmatics. Am Rev Respir Dis 1982;126(3):515-520.

17. Bohadana AB, Peslin R, Hannhart B, Teculescu D. Influence of panting frequency on plethysmographic measurements of thoracic gas volume. J Appl Physiol 1982;52(3):739-747.

18. Habib MP, Engel LA. Influence of the panting technique on the plethysmographic measurement of thoracic gas volume. Am Rev Respir Dis 1978;117(2):265-271.

19. Barter CE, Campbell AH. Comparison of airways resistance measurements during panting and quiet breathing. Respiration 1973;30(1):1-11.

20. Chowienczyk PJ, Rees PJ, Clark TJH. Automated system

for the measurement of airways resistance, lung volumes, and flow-volume loops. Thorax 1981;36(12):944-949.

21. Bates JHT. Correcting for the thermodynamic characteristics of a body plethysmograph. Ann Biomed Eng 1989;17(6):647-655.

22. Lord PW, Brooks AGF. A comparison of manual and automated methods of measuring airway resistance and thoracic gas volume. Thorax 1977;32(1):60-66.

23. Sauder LR. Computer analysis versus technician analysis of body plethysmographic analog recordings of airway resistance and thoracic gas volume. Respir Care 1982;27(1):62-69.

24. Brown R, Hoppin FG Jr, Ingram RH Jr, Saunders NA, McFadden ER Jr. Influence of abdominal gas on the Boyle's law determination of thoracic gas volume. J Appl Physiol 1978;44(3):469-473.

25. Bargeton D, Barres G. Time characteristics and frequency response of body plethysmograph. In: DuBois AB, Van de Woestijne KP, Herzog H, editors. Progress in respiration research, body plethysmography. New York: S Karger; 1969.

26. Crapo RO, Morris AH, Clayton PD, Nixon CR. Lung volumes in healthy nonsmoking adults. Bull Eur Physiopathol Respir 1982;18(3):419-425.

27. NCCLS, GP26-A. A quality system model for health care: approved guideline (1999). Available from NCCLS: phone 610-688-0100; fax 610-688-0700; e-mail exoffice@nccls.org.

28. NCCLS, HS4-A. A quality system model for respiratory care: approved guideline (2000). Available from NCCLS: phone 610-688-0100; fax 610-688-0700; e-mail exoffice@nccls.org.

29. Gardner RM, Clausen JL, Crapo RO, Epler GR, Hankinson JL, Johnson JL, Plummer AL. Quality assurance in pulmonary function laboratories. Am Rev Respir Dis 1986;134(3):625-627.

30. Quanjer PH, Tammeling GJ, Cotes JE, Pedersen OF, Peslin R, Yernault JC. Lung volumes and forced ventilatory flows. Report Working Party Standardization of Lung Function Tests, European Community for Steel and Coal. Official Statement of the European Respiratory Society. Eur Respir J Suppl 1993 Mar;16:5-40.

31. American Thoracic Society. Standardization of spirometry, 1994 update. Am J Respir Crit Care Med 1995;152(3):1107-1136.

32. American Thoracic Society. Pulmonary function laboratory management and procedure manual. Wanger J, editor. New York: ATS; 1998.

33. Kanner RE, Morris AH, Crapo RO, Gardner RM, editors. Clinical pulmonary function testing: a manual of uniform laboratory procedures for the intermountain area, 2nd ed. Salt Lake City: Intermountain Thoracic Society; 1984.

34. Ruppel GL. Manual of pulmonary function testing, 7th ed. St Louis: Mosby-Yearbook; 1998.

35. Demizio DL, Allen PD, Beaudry PH, Coates AL. An alternate method for the determination of functional residual capacity in a plethysmograph. Am Rev Respir Dis 1988;137(2):273-276.

36. Gibson GJ. Measurement of the mechanical properties of the thorax, lungs, and airways. In: Laszlo G, Sudlow MF, editors. Measurement in clinical respiratory physiology. New York: Academic Press; 1982.

37. Recommendations of British Thoracic Society and the Association of Respiratory Technicians and Physiologists. Guidelines for the measurement of respiratory function. Respir Med 1994;88(3):165-194.

38. Zarins LP, Clausen JH. Plethysmography. In: Clausen JL, editor. Pulmonary function testing: guidelines and controversies. New York: Academic Press; 1982.

39. Pelzer A, Thomson ML. Effect of age, sex, stature, and smoking habits on human airway conductance. J Appl Physiol 1966;21(2):469-476.

40. NCCLS, C28-A2. How to define and determine reference intervals in the clinical laboratory: approved guideline, 2nd ed, 2000. Available from NCCLS: phone 610-688-0100; fax 610-688-0700; e-mail exoffice@nccls.org.

41. Gardner RM, Clausen JL, Epler G, Hankinson JL, Permutt S, Plummer AL. Pulmonary function laboratory personnel qualifications. American Thoracic Society Position Paper. ATS News, November 1982.

42. Garner JS. Hospital Infection Control Practices Advisory, Centers for Disease Control and Prevention. Guideline for isolation precautions in hospitals. Am J Infect Control 1996;24(1) or http://www.apic.org/html/resc/gdisolat.html.

43. Centers for Disease Control and Prevention, Hospital Infection Control Practices Advisory Committee. Guideline for infection control in health care personnel, 1998. Am J Infect Control 1998;26:269-354 or Infect Control Hosp Epidemiol 1998;19(6):407-463.

44. Larson EL. APIC guideline for handwashing and hand antisepsis in health care settings. Am J Infect Control 1995;23(4):251-269.

45. Centers for Disease Control & Prevention. Guidelines for preventing the transmission of tuberculosis in health-care facilities, 1994. MMWR 1994;43(RR-13):1-32 or Federal Register 1994;59(208):54242-54303 or http://aepo-xdv-www.epo.cdc.gov/wonder/prevguid/m0035909/m0035909.htm.

46. Rutala WA. APIC guideline for selection and use of disinfectants. Am J Infect Control 1990;18(2):99-117.

47. Kirk YL, Kenday K, Ashworth HA, Hunter PR. Laboratory evaluation of a filter for the control of cross-infection during pulmonary function testing. J Hosp Infect 1992;20:193-198.

ADDITIONAL READING

Coates AL, Peslin R, Rodenstein D, Stocks J. ERS/ATS Workshop Report Series. Measurement of lung volumes by plethysmography. Eur Respir J 1997;10:1415-1427.

AARC Clinical Practice Guideline

Exercise Testing for Evaluation of Hypoxemia and/or Desaturation:
2001 Revision & Update

ETD 1.0 PROCEDURE:
Exercise testing for evaluation of hypoxemia and/or desaturation.

ETD 2.0 DESCRIPTION/DEFINITION:
Exercise testing may be performed to determine the degree of oxygen desaturation and/or hypoxemia that occurs on exertion. Desaturation is defined as a valid decrease in arterial oxygenation as measured by CO-oximetry saturation, (S_{aO_2}) of 2% (based on the reproducibility of HbO_2 measurement at ±1%),[1] an $S_{aO_2} < 88\%$,[2,3] and/or a blood gas $P_{aO_2} \leq 55$ torr.[4]

2.1 Exercise testing may also be performed to optimize titration of supplemental oxygen for the correction of hypoxemia. An S_{pO_2} of 93% should be used as a target.[3]

2.2 It is preferable that this procedure be performed using a method that allows quantitation of workload and heart rate achieved (as % predicted).

   2.2.1 This evaluation can be incorporated into other more complex test protocols (eg, cardiac stress testing).

   2.2.2 Continuous noninvasive measurement of arterial oxyhemoglobin saturation by pulse oximetry can provide qualitative information and an approximation of oxyhemoglobin saturation, with a 4% decrease in S_{pO_2} considered significant,[4] but evaluation of desaturation on exertion requires analysis of arterial blood samples drawn with the subject at rest and at peak exercise.[3,5-12]

2.3 Arterial blood specimens may be obtained by single puncture or by arterial cannulation.[13,14]

2.4 Exercise testing performed with exhaled gas analysis is addressed in a separate guideline.

2.5 This guideline is appropriate for pediatric, adult, and geriatric patients who are capable of following test instructions and techniques.

   2.5.1 The learning ability and communi-

cation skills of the patient being served, should be taken into consideration when performing these tests.

   2.5.2 The neonatal population is not served by this guideline.

ETD 3.0 SETTINGS:
Exercise testing may be performed by trained personnel in a variety of settings including

   3.1 pulmonary function laboratories
   3.2 cardiopulmonary exercise laboratories
   3.3 clinics
   3.4 pulmonary rehabilitation facilities
   3.5 physicians' offices

ETD 4.0 INDICATIONS:
Indications for exercise testing include

   4.1 the need to assess and quantify the adequacy of arterial oxyhemoglobin saturation during exercise in patients who are clinically suspected of desaturation (eg, those who manifest dyspnea on exertion, decreased D_{LCO}, decreased P_{aO_2} at rest, or documented pulmonary disease);[2,7,15-18]

   4.2 the need to quantitate the response to therapeutic intervention (eg, oxygen prescription, medications, smoking cessation, or to reassess the need for continued supplemental oxygen);[2,7,15,19-21]

   4.3 the need to titrate the optimal amount of supplemental oxygen to treat hypoxemia or desaturation during activity;[2,7,21,22]

   4.4 the need for preoperative assessment for lung resection or transplant;[23]

   4.5 the need to assess the degree of impairment for disability evaluation (eg, pneumoconiosis, asbestosis).[24]

ETD 5.0 CONTRAINDICATIONS:
   5.1 Absolute contraindications include

     5.1.1 acute electrocardiographic changes

suggesting myocardial ischemia or serious cardiac dysrhythmias including bradydysrhythmias, tachydysrhythmias, sick sinus syndrome, and multifocal premature ventricular contractions (PVCs), causing symptoms or hemodynamic compromise (occasional PVCs are not a contraindication);[25,29]

5.1.2 unstable angina;[24,25,27]

5.1.3 recent myocardial infarction (within the previous 4 weeks) or myocarditis;[25,26]

5.1.4 aneurysm of the heart or aorta;[25,26]

5.1.5 uncontrolled systemic hypertension;[25,26]

5.1.6 acute thrombophlebitis or deep venous thrombosis;[25,26]

5.1.7 second- or third-degree heart block;[25,26]

5.1.8 recent systemic or pulmonary embolus;[25,26]

5.1.9 acute pericarditis;[25,26]

5.1.10 symptomatic severe aortic stenosis;

5.1.11 uncontrolled heart failure;[25]

5.1.12 uncontrolled or untreated asthma;

5.1.13 pulmonary edema;[25]

5.1.14 respiratory failure;[25]

5.1.15 acute non-cardiopulmonary disorders affected by exercise.

5.2 Relative contraindications include

5.2.1 situations in which pulse oximetry may provide invalid data (eg, elevated HbCO, HbMet, or decreased perfusion). (See AARC Pulse Oximetry Guidelines.[30])

5.2.2 situations in which arterial puncture and/or arterial cannulation may be contraindicated;[31,32]

5.2.3 a non-compliant patient or one who is not capable of performing the test because of weakness, pain, fever, dyspnea, incoordination, or psychosis;[25,26]

5.2.4 severe pulmonary hypertension (cor pulmonale);[25,26]

5.2.5 known electrolyte disturbances (hypokalemia, hypomagnesemia);[25,26]

5.2.6 resting diastolic blood pressure > 110 torr or resting systolic blood pressure > 200 torr;[25,26]

5.2.7 neuromuscular, musculoskeletal, or rheumatoid disorders that are exacerbated

by exercise;[5,26]

5.2.8 uncontrolled metabolic disease (eg, diabetes, thyrotoxicosis, or myxedema;[25,26]

5.2.9 S_{aO_2} or S_{pO_2} < 85% on room air;[5]

5.2.10 complicated or advanced pregnancy;[25]

5.2.11 hypertrophic cardiomyopathy or other forms of outflow tract obstruction;[26]

5.2.12 patient's inability to cooperate or follow directions for testing.

ETD 6.0 PRECAUTIONS AND/OR POSSIBLE COMPLICATIONS:

6.1 Indications for immediate termination of testing include

6.1.1 electrocardiographic abnormalities (eg, dangerous dysrhythmias, ventricular tachycardia, ST-T wave changes);[25,26]

6.1.2 severe desaturation as indicated by an S_{aO_2} ≤80% or S_{pO_2} ≤83% (A number of pulse oximeters have been found to overestimate S_{pO_2};[3,12,33-36]) and/or a 10% fall from baseline values; (Underestimation of saturation has been noted to occur with certain pulse oximeter models.[33,34])

6.1.3 angina;[25,26]

6.1.4 hypotensive responses;

    6.1.4.1 a fall of > 20 torr in systolic pressure, occurring after the normal exercise rise;[37]

    6.1.4.2 a fall in systolic blood pressure below the pre-exercise level;[36]

6.1.5 lightheadedness;[25,26]

6.1.6 request from patient to terminate test.

6.2 Abnormal responses that may require discontinuation of exercise include

6.2.1 a rise in systolic blood pressure to > 250 torr or of diastolic pressure to > 120 torr,[25,26] or a rise in systolic pressure of < 20 torr from resting level;

6.2.2 mental confusion or headache;[25,26]

6.2.3 cyanosis;[25,26]

6.2.4 nausea or vomiting;

6.2.5 muscle cramping.[25,26]

6.3 Hazards associated with arterial puncture, arterial cannulation, and pulse oximetry:[30-32] Pulse oximetry is a noninvasive safe procedure, but because of device limitations, false-nega

tive results for hypoxemia[11] and/or false-positive results for normoxemia or hyperoxemia may lead to inappropriate treatment of the patient. Although it is rare, tissue injury may occur at the measuring site as a result of probe misuse, such as pressure sores from prolonged application or electrical shock and burns from the substitution of incompatible probes between instruments.[30,38-42]

ETD 7.0 LIMITATIONS OF PROCEDURE/ VALIDATION OF RESULTS:

7.1 Limitations of equipment:

7.1.1 Because of possible limitations of pulse oximetry with exercise and at rest, measurements may read falsely low or falsely high and should be validated by comparison with baseline arterial samples analyzed by CO-oximetry.[30,33,43]

7.1.1.1 Only a limited number of pulse oximeters have been validated with results of concurrent arterial blood gas analysis in diseased subjects under exercise conditions.[16]

7.1.1.2 Overestimation of oxygen saturation may occur with carboxyhemoglobin saturations (> 4 %).[2,44,45]

7.1.1.3 Decreasing accuracy in S_{pO_2} has been reported with desaturations to < 83%. This is assumed to be the result of limitations of in vivo calibration to 85% with extrapolation of the calibration curve below that value.[13,45]

7.1.1.4 Decreased perfusion with cardiovascular disease, vasoconstriction, or hypothermia may result in false-positive results or no valid data in some pulse oximeter models.[11,46] Use of an alternative site should be evaluated (eg, ear, finger, forehead). Alternative handwarming methods may be used to increase circulation.

7.1.1.5 Reduced ear perfusion associated with heavy exercise has been shown to affect S_{pO_2} in some models of pulse oximeters.[11,47-49]

7.1.1.6 Motion artifact may appear with exercise.[16,50] Some pulse oximeters are better then others at rejecting motion artifact.[51,52]

7.1.1.7 Pulse oximeter response time may be inadequate to describe rapid changes in saturation.[7,16,45]

7.1.1.8 Skin pigmentation should, in theory, not affect pulse oximeter readings, but various studies report conflicting data depending on the manufacturer and model.[3,45]

7.1.1.9 Hemoglobin disorders may affect the accuracy of the pulse oximeter reading.[16,33,45] Important underestimation of arterial saturation may result from pulse oximetry in subjects with total hemoglobin levels of ≤8 g/dL.[53]

7.1.1.10 Pulse oximetry is less useful over the range in which large changes in P_{aO_2} are associated with small changes in S_{aO_2} (ie, P_{aO_2} ≥60 torr).[16]

7.1.1.11 Ambient light during testing may interfere with measurements of pulse oximetry.[45]

7.1.1.12 Exercise testing in which oxyhemoglobin saturation by pulse oximetry is the only variable measured provides limited information.

7.1.2 Limitations related to the patient:

7.1.2.1 Additional limitations common to arterial sampling and analysis under resting conditions should be considered.[31,32]

7.1.2.2 Patient cooperation level or physical condition may limit the subject's ability to exercise at a workload sufficient to evoke a response.[25,26] Variables that are not adequately monitored (eg, free walking) have limited application.

7.2 Validation of results:

7.2.1 Arterial blood gas samples should be obtained at rest and at peak exercise. Samples from single arterial punctures have been shown to be equivalent to samples drawn from indwelling cannulas.[15,54]

7.2.2 In the unlikely event that a single puncture at peak exercise is unsuccessful in an uncannulated patient, a sample drawn within 10-15 seconds of the termination of exercise will suffice unless analysis shows a decrease from the resting values, in which case quantitation of de-

saturation requires a peak exercise sample obtained by cannula.[15]

7.2.3 Arterial blood gas results should be obtained according to the Guidelines for arterial blood gas sampling and for arterial blood gas analysis.[31,32,54]

7.2.4 Validity of pulse oximetry results is verified by comparison with the results of analysis by CO-oximetry,[30,51] preferably at rest and at end of exercise.

7.2.4.1 S_{pO_2} may be used to assess response to supplemental oxygen. If administration of supplemental oxygen does not improve a low S_{pO_2}, arterial blood analysis may be warranted.

7.2.4.2 Testing should be performed in compliance with the AARC Pulse Oximetry Clinical Practice Guideline.[30]

7.2.4.3 Correlation between pulse oximetry heart rate and palpated pulse rate and/or electrocardiogram should be established.[45]

7.2.4.4 Pulse oximetry with pulse waveform display may be desirable. For patients with normal adult hemoglobin, the highest accuracy and best performance is attained when the probe is attached to the patient in such a way that the arterial signal has the largest possible amplitude, which is only available with systems that yield a plethysmographic tracing.[45]

ETD 8.0 ASSESSMENT OF NEED:

Exercise testing for evaluation of hypoxemia and/or desaturation may be indicated (see section ETD 4.0 INDICATIONS) in the presence of

8.1 a history and physical indicators suggesting hypoxemia and/or desaturation (eg, dyspnea, pulmonary disease);

8.2 abnormal diagnostic test results (eg, D_{LCO}, FEV_1, resting arterial blood gases including directly measured HbO_2, HbCO, and HbMet);

8.3 the need to titrate or adjust a therapy (eg, supplemental oxygen).

ETD 9.0 ASSESSMENT OF QUALITY OF TEST AND VALIDITY OF RESULTS:

The consensus of the committee is that all diagnostic procedures should follow the quality model described in the NCCLS GP26-A A Quality System Model for Health Care.[55] (Fig. 1) The document describes a laboratory path of workflow model that incorporates all the steps of the procedure. This process begins with patient assessment and the generation of a clinical indication for testing through the application of the test results to patient care. The quality system essentials defined for all health care services provide the framework for managing the path of workflow. A continuation of this model for respiratory care services is further described in NCCLS HS4-A A Quality System Model for Respiratory Care.[56] In both quality models the patient is the central focus.
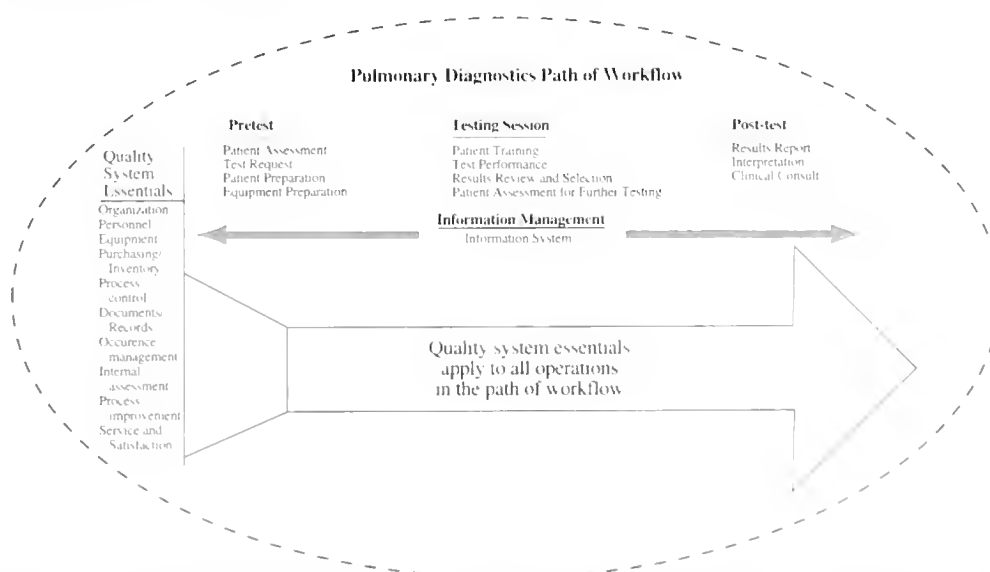
Fig. 1. Structure for a Quality System Model for a Pulmonary Diagnostics Service (From Reference 55, with permission)

9.1 General considerations include:

9.1.1 As part of any quality assurance program, indicators must be developed to monitor areas addressed in the path of workflow.

9.1.2 Each laboratory should standardize procedures and demonstrate intertechnologist reliability. Test results can be considered valid only if they are derived according to and conform to established laboratory quality control, quality assurance, and monitoring protocols.

9.1.3 Documentation of results, therapeutic intervention (or lack of) and/or clinical decisions based on the exercise testing should be placed in the patient's medical record.

Report of test results should contain a statement by the technician performing the test regarding test quality (including patient understanding of directions and effort expended) and, if appropriate, which recommendations were not met.

9.1.4 The type of medications, dose, and time taken prior to testing and the results of the pretest assessment should be documented.

9.1.5 Test results should be interpreted by a physician, taking into consideration the clinical question to be answered.[57]

9.1.6 A technologist who has not met annual competency requirements or whose competency is deemed unacceptable as documented in an occurrence report should not be allowed to participate, until he has received remedial instruction and has been re-evaluated.

9.1.7 There must be evidence of active review of quality control, proficiency testing, and physician alert, or 'panic' values, on a level commensurate with the number of tests performed

9.2 Calibration and quality control measures specific to equipment used in exercise testing for desaturation include:

9.2.1 Calibration procedures as defined by the laboratory protocols and manufacturer's specifications should be adhered to.[42]

9.2.2 Treadmills and bicycle ergometers should be calibrated according to the manufacturer's recommendations, with periodic re-verification. (One reference suggests every 3-6 months.[42])

9.2.3 Pulse oximeters monitors should be maintained as described under quality assurance in the manufacturer's manual.

9.2.4 Biological controls should be tested regularly (self-testing of normal laboratory staff).[58]

9.3 Test quality: Results of arterial blood gas analysis and/or S_{pO_2} should confirm or rule out oxygen desaturation during exercise to validate the patient's clinical condition.

9.4 Test results: The exercise should have a symptom-limited or physiologic end point documented (eg, heart rate or onset of dyspnea).

ETD 10.0 RESOURCES:

10.1 Equipment:

10.1.1 Treadmill, cycle ergometer, or equivalent equipment, adaptable to patients who may be severely limited (eg, low-speed treadmill, low-watt ergometer, arm crank ergometer).[25,26,59-61] Other forms of exercise may be utilized (stair climbing, step test, timed walking); however, such modes do not eliminate the necessity for adequate monitoring as described in Sections 7 and 9 and the necessity for adequate documentation of procedure and patient response.

10.1.2 Arterial blood sampling equipment for single puncture or arterial cannulation and analyzers that have been properly calibrated and for which multilevel controls indicate proper function[31,32,54]

10.1.3 Pulse oximeter monitor and related accessories.[30]

10.1.4 Electrocardiographic monitor with the capacity to monitor heart rate to a predicted maximum and accurately display cardiac rhythm during exercise. (Multiple leads are preferred.)[25,26]

10.1.5 Resuscitation equipment including oxygen with various delivery devices, such as nasal cannula and mask.[25,26]

10.1.6 An easily accessible cardiac arrest cart and defibrillator with resuscitation

equipment[25,26,59]

10.1.7 Blood pressure monitoring device, manual or automatic. (If an automated system is used, a manual blood pressure cuff and stethoscope should be available as a backup.)[25,26]

10.1.8 Visual aids (eg, Borg scales for dyspnea and fatigue) that are large, easy to read, and in clear view.[59,62,63]

10.1.9 Blood gas sampling and analysis equipment.[31,32,54]

10.2 Background history and data:

10.2.1 Results of appropriate baseline diagnostic tests and patient history (eg, electrocardiogram, chest radiograph, and pulmonary function test results) should be available.[25,26,29]

10.2.2 The need for written consent should be determined within the specific institution.[26,27]

10.2.3 A list of the patient's current medications and any pharmacologic allergies should be included.

10.3 Personnel:

10.3.1 The presence of a physician trained in exercise testing may be required depending on patient condition and hospital policy.[25,26,59]

10.3.2 Personnel administering the test should possess experience and knowledge in exercise physiology and testing, including arterial blood gas sampling and analysis; cardiopulmonary resuscitation (certified in Basic Cardiac Life Support, or BCLS. Qualification in Advanced Cardiac Life Support, or ACLS, is recommended); ECG abnormality recognition; oxygen therapy; blood pressure monitoring; and application and limitations of pulse oximeters.[28] Training and demonstrated competency must be documented for all testing personnel.[56]

10.3.3 Testing personnel should have the knowledge and skills to respond to adverse situations with the patient and to know when cessation of further testing is indicated (versus coaching the patient to continue).[28,57-59]

ETD 11.0 MONITORING:

11.1 Recommended monitoring of patient during testing:

11.1.1 Electrocardiograph with strip recorder, preferably screened in real-time to check for displaced leads.

11.1.2 Oxygen delivery devices with documented F_{DO_2}

11.1.3 Physical assessment (chest pain, leg cramps, color, perceived exertion, dyspnea)[25,26]

11.1.4 Respiratory rate[25,26]

11.1.5 Patient cooperation and effort level

11.1.6 Borg, modified Borg, or visual analog dyspnea or symptom scales[64,65]

11.1.7 Blood gas sampling using site and technique consistent with the AARC Clinical Practice Guideline for blood gas sampling,[31] and NCCLS Guidelines[54]

11.1.8 Continuous monitoring of oxygenation status (S_{pO_2})

11.1.9 Heart rate, rhythm, and ST-T wave changes[25,26]

11.1.10 Blood pressure[25,26]

11.2 Recommended equipment monitoring during testing: Pulse waveforms of S_{pO_2} and/or S_{aO_2} should be analyzed to assure adequate signal acquisition for reliable readings.

ETD 12.0 FREQUENCY:

The frequency of testing depends on the patient's clinical condition and the need for changes in therapy. Exercise may be repeated for certification of supplemental oxygen needs.

ETD 13.0 INFECTION CONTROL:

13.1 The staff, supervisors, and physician-directors associated with the pulmonary laboratory should be conversant with "Guideline for Isolation Precautions in Hospitals" made by the Centers for Disease Control and the Hospital Infection Control Practices Advisory Committee (HICPAC),[66] and develop and implement policies and procedures for the laboratory that comply with its recommendations for Standard Precautions and Transmission-Based Precautions.

13.2. The laboratory's manager and its medical director should maintain communication and

cooperation with the institution's infection control service and the personnel health service to help assure consistency and thoroughness in complying with the institution's policies related to immunizations, post-exposure prophylaxis, and job- and community-related illnesses and exposures.[67]

13.3 Primary considerations include:

13.3.1 adequate handwashing.[68]

13.3.2 provision of prescribed ventilation with adequate air exchanges,[69]

13.3.3 careful handling and thorough cleaning and processing of equipment.[66] Procedure-specific considerations include:

13.3.3.1 disposable items are for single patient use;

13.3.3.2 disposable electrodes should be used for electrocardiographic monitoring with Standard Precautions observed during patient skin preparation. Cables and equipment that touch the patient should be wiped down with a disinfectant after each use;

13.3.3.3 reusable pulse oximeter probes should be cleaned between patient use, following the manufacturer's guidelines.

13.3.4 the exercise of particular care in scheduling and interfacing with the patient in whom a diagnosis has not been established.

ETD 14.0 AGE SPECIFIC ISSUES

14.1 This guideline does not apply to the neonatal population.

14.2 This CPG document applies to pediatric, adolescent, adult, and geriatric populations.

14.3 Test instructions and techniques should be given in a manner that takes into consideration the learning ability, communication skills, and age of the patient being served.

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REFERENCES

1. Mohler JG, Collier CR, Brandt W, Abramson J, Kerkaik G, Yates S. Blood gases. In: Clausen JL, editor. Pulmonary function testing guidelines and controversies: equipment, methods, and normal values. New York: Academic Press; 1982: 251.

2. American Thoracic Society. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease: official statement. Am J Respir Crit Care Med 152(3):S77-S120.

3. Carone M, Patessio A, Appendini L, Purro A, Czernicka E, Zanaboni S, Donner CF. Comparison of invasive and noninvasive saturation monitoring in prescribing oxygen during exercise in COPD patients. Eur Respir J. 1997;10(2):446-451.

4. Tobin M. Respiratory monitoring. JAMA 1990;264:244-251.

5. Zeballos RJ, Weisman IM. Reliability of noninvasive oximetry in black subjects during exercise and hypoxia. Am Rev Respir Dis 1991;144:1240-1244.

6. Schnapp LM, Cohen NH. Pulse oximetry uses and abuses. Chest 1990;98(5):1244-1250.

7. Escourrou PJL, Delaperche MF, Visseaux A. Reliability of pulse oximetry during exercise in pulmonary patients. Chest 1990;97(3):635-638.

8. Powers SK, Dodd S, Freeman J, Ayers GD, Samson H, McKnight T. Accuracy of pulse oximetry to estimate HbO$_2$ fraction of total Hb during exercise. J Appl Physiol 1989;67(1):300-304.

9. Douglas NJ, Brash HM, Wraith PK, Calverley PM, Leggett RJ, McElderry L, Flenley DC. Accuracy sensitivity to carboxyhemoglobin, and speed of response of the Hewlett-Packard 47201A ear oximeter. Am Rev Respir Dis 1979;119(2):311-313.

10. Hansen JE, Casaburi R. Validity of ear oximetry in clinical exercise testing. Chest 1987;91(3):333-337.

11. Powers SK, Dodd S, Woodyard J, Beadle RE. Haemoglobin saturation during incremental arm and leg exercise. Br J Sports Med 1984;18(3):212-216.

12. Smyth RJ, D'Urzo AD, Slutsky AS, Galko BM, Rebuck AS. Ear oximetry during combined hypoxia and exercise. J Appl Physiol 1986;60(2):716-719.

13. Ries AL, Fedullo PF, Clausen JL. Rapid changes in arterial blood gas levels after exercise in pulmonary patients. Chest 1983;83(3):454-56.

14. Frye M, DiBenedetto R, Lain D, Morgan K. Single arterial

puncture vs arterial cannula for arterial gas analysis after exercise. Chest 1988;93(2):294-298.

15. Ries AL, Farrow JT, Clausen JL. Accuracy of two ear oximeters at rest and during exercise on pulmonary patients. Am Rev Respir Dis 1985;32:685-689.

16. Clark JS, Votteri B, Ariagno RL, Cheung P, Eichhorn JH, Fallat RJ, et al. Noninvasive assessment of blood gases. Am Rev Respir Dis 1992;145(1):220-232.

17. Fukushima T, Ohrui T, Itabashi S, Sekizawa K, Arkawa T, Yanai M, et al. Prolonged hypoxemia after 10 minutes walking exercise in aged patients with chronic obstructive pulmonary disease. Tohoku J Exp Med 1990;162(4):345-353.

18. Ries AL, Farrow JT, Clausen LF. Pulmonary function tests cannot predict exercise-induced hypoxemia in COPD. Chest 1988;93:454-459.

19. Moore DP, Weston AR, Hughes JMB, Oakley CM, Cleland JGF. Effects of increased inspired oxygen concentrations on exercise performance in chronic heart failure. Lancet 1992;339:850-853.

20. Carlone S, Angelici E, Palange P, Serra P, Farber MO. Effects of fenoterol on oxygen transport in patients with chronic airflow obstruction. Chest 1988;93(4):790-794.

21. Bower JS, Brook CJ, Zimmer K, Davis D. Performance of a demand oxygen saver system during rest, exercise, and sleep in hypoxemic patients. Chest 1988;94(1):77-80.

22. Couser JI Jr, Make BJ. Transtracheal oxygen decreases inspired minute ventilation. Am Rev Respir Dis 1989;139(3):627-631.

23. Morice RC, Peters EJ, Ryan MB, Putnam JB, Ali MK, Roth JA. Exercise testing in the evaluation of patients at high risk for complications from lung resection. Chest 1992;101:356-361.

24. Britton MG, Apps MCP, Maxwell DL, Hughes DTD, Hanson A. The value of ear lobe oximetry in the assessment of disability in asbestos-related disease. Respir Med 1989;83:43-49.

25. American College of Sports Medicine. Guidelines for graded exercise testing and exercise prescription, 5th ed. Pennsylvania: Williams & Wilkins; 1995.

26. Gibbons RJ, Balady GJ, Beasley JW, Bricker JT, Duvernoy WF, Froelicher VF, et al. ACC/AHA Guidelines for Exercise Testing: A report of the American College of Cardiology and the American Heart Association Task Force on Practice Guidelines (Committee on Exercise Testing). J Am Coll Cardiol 1997;30(1):260-311.

27. Gibbons RJ, Balady GJ, Beasley JW, Bricker JT, Duvernoy WF, Froelicher VF, et al. ACC/AHA guidelines for exercise testing: executive summary, a report of the American College of Cardiology and the American Heart Association Task Force on Practice Guidelines (Committee on Exercise Testing). ACC/AHA Practice Guidelines. Circulation 1997;96(1):345-354.

28. Pina IL, Balady GJ, Hanson P, Labovitz AJ, Madonna DW, Myers J. Guidelines for clinical exercise testing laboratories. A statement for healthcare professionals from the Committee on Exercise and Cardiac Rehabilitation, American Heart Association. Circulation 1995;91(3):912-921.

29. Wasserman K, Hansen JE, Sue DY, Casaburi R, Whipp BJ.

Principles of exercise testing and interpretation, 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 1999.

30. American Association for Respiratory Care. AARC Clinical Practice Guideline: Pulse oximetry. Respir Care 1991;36(12):1046-1048.

31. American Association for Respiratory Care. AARC Clinical Practice Guideline: Sampling for arterial blood gas analysis. Respir Care 1992;37(8):913-917.

32. American Association for Respiratory Care. AARC Clinical Practice Guideline. Arterial blood analysis 2001 revision & update. Respir Care 2001;46(5):498-505.

33. Wood RJ, Gore CJ, Hahn AG, Norton KI, Scroop GC, Campbell DP, et al. Accuracy of two pulse oximeters during maximal cycling exercise. Austral J Sci Med in Sport 1997;29(2):47-50.

34. Norton LH, Squires B, Craig P, McLeay G, McGrath P, Norton KI. Accuracy of pulse oximetry during exercise stress testing. Int J Sports Med 1992;13(7):523-527.

35. Severinghaus JW, Naifeh KH. Accuracy of response of six pulse oximeters to profound hypoxia. Anesthesiology 1987;67:551-558.

36. Severinghaus JW, Naifeh KH, Koh SO. Errors in 14 pulse oximeters during profound hypoxia. J Clin Monit 1989;5:72-81.

37. Dubach P, Froelicher VF, Klein J, Oakes D, Grover-McKay M, Friis R. Exercise-induced hypotension in a male population. Circulation 1988;78:1380-1387.

38. Hannhart B, Michalski H, Delorme N, Chapparo G, Polu J-M. Reliability of six pulse oximeters in chronic obstructive pulmonary disease. Chest 1991;99:842-846.

39. Tremper KK, Barker SJ. Pulse oximetry. Anesthesiology 1998;70:98-108.

40. Praud JP, Carofilis A, Bridley F, Dehan M, Gaultier CL. Accuracy of two wavelength pulse oximetry in neonates and infants. Pediatr Pulmonol 1989;6:180-182.

41. ECRI Health Devices Alert, 1990-A26. Plymouth Meeting PA. ECRI, June 29, 1990.

42. Wanger J, editor. ATS pulmonary function laboratory management and procedure manual. New York: American Thoracic Society; 1998; Chapters 17,18.

43. Jensen LA, , Onyskiw JE, Prasad NGN. Meta-analysis of arterial oxygen saturation monitoring by pulse oximetry in adults. Heart Lung 1998;27(6):387-408.

44. Tweeddale P, Douglas N. Evaluation of Biox IIa ear oximeter. Thorax 1985;40:825-827.

45. Moyle JTB. Principles and practice series: pulse oximetry. London: BMJ Books; 1998.

46. Morris RW, Nairn M, Torda TA. A comparison of fifteen pulse oximeters. Part I: a clinical comparison; Part II: a test of performance under conditions of poor perfusion. Anaesth Intensive Care 1989;17:62-82.

47. Williams J, Powers S, Stuart M. Hemoglobin desaturation in highly trained endurance athletes during heavy exercise. Med Sci Sports Exerc 1986;18:168-173.

48. Godfrey S, Wozniak ER, Courtney-Evans RJ, Samuels CS. Ear lobe blood samples for blood gas analysis at rest and during exercise. Br J Dis Chest 1971;65:58-65.

49. Martin D, Powers S, Cicale M, Collop N, Huang D, Criswell D. Validity of pulse oximetry during exercise in

elite endurance athletes. J Appl Physiol 1992;72(2):455-458.

50. Warley AH, Mitchell JH, Stradling JR. Evaluation of the Ohmeda 3700 pulse oximeter. Thorax 1987;42:892-896.

51. Blonshine S. Pulse oximetry technology advances. AARC Times 1999;23(2):45-48,89.

52. Poets CF, Stebbens VA. Detection of movement artifact in recorded pulse oximeter saturation. Eur J Pediatr 1997;156(10):808-811.

53. Severinghaus JW, Koh SO. Effect of anemia on pulse oximeter accuracy at low saturation. J Clin Monit 1990(6):85-88.

54. Blonshine S, Alberti R, Olesinski RL. NCCLS H11–A3, 3rd ed. Procedures for the collection of arterial blood specimens: approved standard. May 1999;19(8). Available from NCCLS: phone 610-688-0100; Fax 610-688-0700; e-mail exoffice@nccls.org.

55. NCCLS. GP26-A A quality system model for health care: approved guideline (1999). Available from NCCLS: phone 610-688-0100; Fax 610-688-0700; e-mail exoffice@nccls.org.

56. NCCLS. HS4-A A quality system model for respiratory care. Available from NCCLS: phone 610-688-0100; Fax 610-688-0700; e-mail exoffice@nccls.org.

57. European Respiratory Society. Clinical exercise testing with reference to lung diseases: indications, standardization and interpretation strategies. ERS Task Force on Standardization of Clinical Exercise Testing. Eur Respir J 1997;10(11):2662-2689.

58. Morgan MDL. Exercise testing. In: Hughes JMB, Pride NB editors. Lung function tests: physiological principles and clinical applications. Philadelphia: WB Saunders; 1999.

59. Lear SA, Brozic A, Myers JN, Ignaszewski A. Exercise stress testing: an overview of current guidelines Sports Med 1999; May 27 (5):285–312.

60. Martin TW, Zeballos RJ, Weisman IM. Use of arm crank exercise in the detection of abnormal pulmonary gas exchange in patients at low altitude. Chest 1992;102:169-175.

61. Martin TW, Zeballos RJ, Weisman IM. Gas exchange during maximal upper extremity exercise. Chest 1991;99:420-425.

62. Borg GAV. Psycho-physical basis of perceived exertion. Med Sci Sports Exercise 1982;14:377–381.

63. Borg GA. Borg's perceived exertion and pain scales. Champaign: Human Kinetics; 1998

64. Stephens JM. Walking speed on parquetry and carpet after stroke: effect of surface and retest reliability. Clin Rehabil 1999;13:171-181.

65. Aitken RCB. Measurement of feelings using visual analogue scales. Proc R Soc Med 1969;62:989–993.

66. Garner JS. Hospital Infection Control Practices Advisory Committee, Centers for Disease Control and Prevention. Guidelines for isolation precautions in hospitals. Atlanta GA: Centers for Disease Control and Prevention, 1-01-1996. www.cdc.gov or Am J Infect Control 1996;24(1):24-52.

67. Centers for Disease Control and Prevention, Hospital Infection Control Practices Advisory Committee. Guideline for infection control in health care personnel, 1998. Am J Infect Control 1998;26:269-354 or Infect Control Hosp Epidemiol 1998;19:407-463.

68. Larson EL. APIC guideline for handwashing and hand antisepsis in health care settings. Am J Infect Control 1995;23(4):251-269

69. Centers for Disease Control & Prevention. Guidelines for preventing the transmission of tuberculosis in health-care facilities. 1994. MMWR 1994;43(RR-13):1-32 or Federal Register 1994;59(208):54242-54303 or http://aepo-xdv-www.epo.cdc.gov/wonder/prevguid/m0035909/m0035909.htm.

ADDITIONAL READING

Jones NL. Clinical exercise testing, 4th ed. Philadelphia: WB Saunders Co; 1997.

Zavala D. Manual on exercise testing, 2nd ed. Iowa City: University of Iowa Press; 1987.

Fletcher GF, Balady G, Froelicher VF, Hartley LH, Haskell WL, Pollock ML. Exercise standards: a statement for health professionals from the American Heart Association. Special Report. Circulation 1995;91:580-615.

Astrand PO, Rodahl K. Textbook of work physiology, 2nd ed. McGraw-Hill; 1977.

Ellestad MH, Blomquist CG, Naughton JP. Standards for adult exercise testing laboratories. Circulation 1979;59 (Suppl):421A-430A.

AARC Clinical Practice Guideline

Methacholine Challenge Testing: 2001 Revision & Update

MCT 1.0 PROCEDURE:

Methacholine challenge test. This guideline does not address other bronchial challenges (eg, histamine, exercise, occupational exposures, specific antigens, isocapnic hyperventilation.)

MCT 2.0 DESCRIPTION/DEFINITION:

2.1 The methacholine challenge test is one method of assessing airway responsiveness. In this test, the patient inhales an aerosol of one or more concentrations of methacholine. Results of pulmonary function tests (eg, spirometry, specific conductance) performed before and after the inhalations are used to quantitate response. This guideline applies to adults and children capable of adequately performing spirometry or body plethysmography and of cooperating during the course of the challenge.

2.2 A positive test is defined as a decrease from the baseline forced expiratory volume in the first second (FEV_1) or of the postdiluent FEV_1 value of 20%, or of a decrease in specific conductance of 35-45% from the baseline or postdiluent value.[1-4]

MCT 3.0 SETTINGS:

Possible settings include:

3.1 pulmonary function laboratory;

3.2 clinic or physician's office;

3.3 field site (eg, occupational setting or workplace).

MCT 4.0 INDICATIONS:

Indications for testing include:

4.1 the need to exclude a diagnosis of airway hyperreactivity (ie, asthma);[1,2,5-9]

4.2 the need to evaluate occupational asthma;[1,2]

4.3 the need to assess the severity of hyperresponsiveness;[1,2]

4.4 the need to determine the relative risk of developing asthma;[2]

4.5 the need to assess response to therapeutic interventions;[2]

MCT 5.0 CONTRAINDICATIONS:

5.1 Absolute contraindications are:

5.1.1 ventilatory impairment: FEV_1 <50% of predicted or < 1.0 L;[2] [This may be a relative contraindication depending on the age or size of the patient or on the presence of a restrictive lung disorder (reduced forced vital capacity, or FVC, with a relatively normal FEV_1/FVC)];

5.1.2 heart attack or stroke within the previous 3 months;[1,2]

5.1.3 known aortic or cerebral aneurysm;[1,2]

5.1.4 uncontrolled hypertension [The American Thoracic Society (ATS) suggests systolic pressure > 200 and/or diastolic pressure >110 mm Hg.];[2]

5.2 Relative contraindications are:

5.2.1 ventilatory impairment: FEV_1 > 50% or > 1.5L but < 60% of predicted;[2]

5.2.2 inability to perform spirometry of acceptable quality;[2]

5.2.3 significant response to the diluent, if administered (ie, > 10% fall in FEV_1 from baseline);[10]

5.2.4 upper- or lower-respiratory-tract infection within previous 2 to 6 weeks;[1,11,12]

5.2.5 current use of cholinesterase-inhibitor medication (for myasthenia gravis);[2]

5.2.6 pregnancy (The effect of methacholine on the fetus is unknown.);[13]

5.2.7 lactation;[13]

5.3 Failure to withhold medications may affect the methacholine challenge test. Recommended periods for withholding medications are generally based on their duration of action.[1,2] Laboratories may choose to develop a simplified withholding schedule that makes allowances for any of the following used by the patient:

Agent	Withholding Time
short-acting inhaled bronchodilators	6-8 hours
long-acting inhaled bronchodilators (eg: salmeterol, formoterol)	48 hours
anticholinergic aerosols (eg: ipratropium)	24 hours
tiotropium	up to 1 week[14]
disodium cromoglycate	8 hours
nedocromil	48 hours
oral beta$_2$-adrenergic agonists	24 hours
theophyllines, depending on specific preparation[2]	12-48 hours
leukotriene modifiers	24 hours[2]
corticosteroids, inhaled or oral (may decrease hyperresponsiveness)	Duration of effect is unknown but may be prolonged.[15,16]

5.4 Foods: Ingestion of coffee, tea, cola drinks, chocolate, or other foods containing caffeine may decrease bronchial responsiveness. These substances should be withheld on the day of test.

5.5 Other factors that may confound results include:

5.5.1 smoking,[17]

5.5.2 occupational sensitizers,[18]

5.5.3 respiratory infection,[11,12]

5.5.4 specific antigens,[19]

5.5.5 vigorous exercise.[20-22] (Performing other bronchial challenge procedures or exercise testing immediately prior to methacholine challenge may affect interpretation.)

MCT 6.0 HAZARDS/COMPLICATIONS:

Possible hazards or untoward reactions include:

6.1 bronchoconstriction, hyperinflation, severe coughing;

6.2 hazards associated with spirometry, such as dizziness, light-headedness, chest pain;[23]

6.3 possible exposure of testing personnel to provocative substance.

MCT 7.0 LIMITATIONS OF METHOD & VALIDATION OF RESULTS:

7.1 Limitations of pulmonary function testing used to quantitate response including intralaboratory variability for each pulmonary function test variable:

7.1.1 In some patients, spirometry may not be sensitive enough or specific enough to detect response, and other measurements such as airways resistance (R_{aw}) and/or specific conductance (sG_{aw}) may be used. Differences of opinion exist regarding the spirometric values that best track response in particular airways.[1,2,24]

7.1.2 Deep inspiration taken while performing spirometry variably alters bronchial tone and may result in either bronchoconstriction or bronchodilatation.[25-27]

7.1.3 Poor patient effort during pulmonary function testing can produce false-positive results and make interpretation more difficult or impossible. Results from spirometry should be acceptable according to the most recent ATS recommendations, and the quality of the flow-volume curves should be examined after each maneuver.[2,28]

7.1.4 Spirometry should be performed according to the current acceptability guidelines of the ATS. Alternatively, the expiratory maneuver can be shortened to about 2 seconds after the methacholine doses are inhaled if FEV_1 is the only outcome measure. If this shortened expiratory maneuver is used, care should be taken to assure that the inspiration is maximal.[2] After the inhalation of diluent (if used) and of each dose of methacholine, FEV_1 measurements should be made at 30 and 90 seconds after the last inhalation. The time interval between doses should be standardized at 5 minutes to keep cumulative effect constant.

7.2 A limitation of the method is the variability due to the effects of various factors including medications, time of day, and differences in technique and equipment.

7.3 Inconsistencies in technique and equipment can affect the amount of agonist reaching the airways and, thus, the subject's response—making meaningful interpretation difficult or impossible. Factors influencing response that must be controlled and held constant across testing include nebulizer output and particle size, volume inhaled, length of breath-hold, and inspiratory flow.[2,29,30]

7.4 If clinical suspicions are not confirmed by

one test, additional tests may be indicated.

7.5 The final test report should include:

7.5.1 $PC_{20}FEV_1$ (ie, the provocative concentration that causes a 20% fall in FEV_1).

7.5.2 comment on the adequacy of spirometric effort and quality of other measurements;

7.5.3 notation regarding medications known to confound interpretation of results (Section 5.3) taken by the patient prior to testing;

7.5.4 presence or absence of other factors known to confound interpretation of results (Section 5.4);

7.5.5 clinical signs and symptoms and clinical appearance during the course of the test and after final dose;

7.5.6 bronchodilator and dose administered at end of challenge;

7.5.7 tabular display of data for each test phase including response to bronchodilator at end of challenge.

MCT 8.0 ASSESSMENT OF NEED:

Need is established by documenting in a subject the presence of one or more of the listed indications or as established by progression through the institution's or the laboratory's protocol decision tree.

MCT 9.0 ASSESSMENT OF TEST QUALITY & VALIDITY OF RESULTS:

The consensus of the committee is that all diagnostic procedures should follow the quality model described in the NCCLS GP26-A A Quality System Model for Health Care.[31] (Fig. 1) The document describes a laboratory path of workflow model that incorporates all the steps of the procedure. This process begins with patient assessment and the generation of a clinical indication for testing through the application of the test results to patient care. The quality system essentials defined for all health care services provide the framework for managing the path of workflow. A continuation of this model for respiratory care services is further described in NCCLS HS4-A A Quality System Model for Respiratory Care.[32] In both quality models the patient is the central focus.

9.1 General considerations include:

9.1.1 As part of any quality assurance program, indicators must be developed to monitor areas addressed in the path of workflow.

9.1.2 Each laboratory should standardize procedures and demonstrate intertechnologist reliability. Test results can be considered valid only if they are derived according to and conform to established laboratory quality control, quality assurance, and monitoring protocols.

9.1.3 Documentation of results, therapeutic intervention (or lack of) and/or clinical decisions should be placed in the patient's medical record.

9.1.4 The type of medications, dose, and time taken prior to testing and the results of the pretest assessment should be docu-
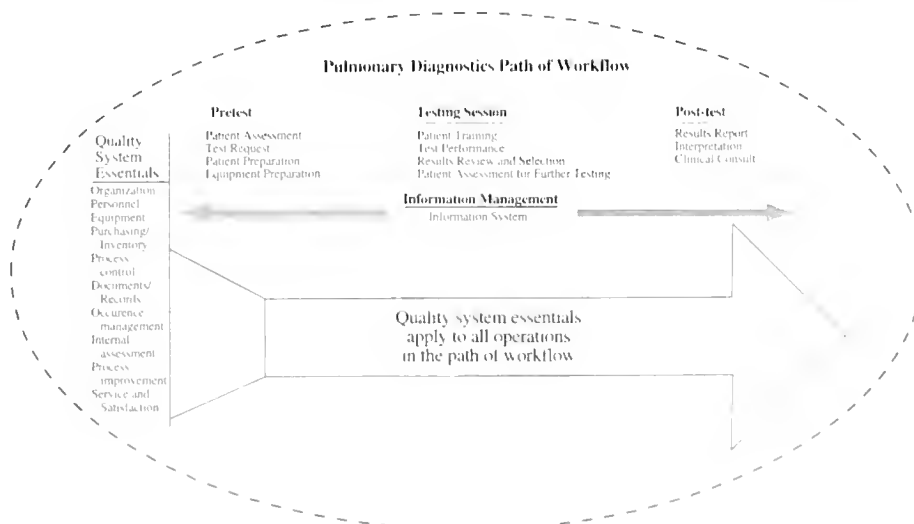
Fig. 1. Structure for a Quality System Model for a Pulmonary Diagnostics Service (From Reference 31, with permission)

mented.

9.1.5 Report of test results should contain a statement by the technician performing the test regarding test quality (including patient understanding of directions and effort expended) and, if appropriate, which recommendations were not met.

9.1.6 Test results should be interpreted by a physician, taking into consideration the clinical question to be answered.

9.1.7 Personnel who do not meet annual competency requirements or whose competency is deemed unacceptable as documented in an occurrence report should not be allowed to participate, until they have received remedial instruction and have been re-evaluated.

9.1.8 There must be evidence of active review of quality control, proficiency testing, and physician alert, or 'panic' values, on a level commensurate with the number of tests performed.

9.2 Calibration and quality control measures specific to equipment used in methacholine challenge include:

9.2.1 the size of the dose received and, thus, the response and its interpretation include nebulizer output, particle size, inspiratory flow, lung volume at beginning of inspiration, and breath-hold time (These factors must be held constant across the testing procedure and from one test to another.);

9.2.2 excessive variability in measured values including a nonreproducible baseline (FEV_1 variation of more than 0.2 L after repeated efforts) makes test results more difficult to interpret.[28]

9.3 Recommendations related to equipment maintenance and calibration made in the Clinical Practice Guidelines for spirometry[23] and measurement of specific conductance[33] should be addressed.

MCT 10.0 RESOURCES:

10.1 Equipment:

10.1.1 Spirometers must meet or exceed ATS requirements[28] and be calibrated appropriately. All other equipment must be appropriately calibrated and maintained.

10.1.2 A high quality nebulizer with consistent output should be used to produce the aerosol. The particles produced by the nebulizer should have a mass median aerodynamic diameter (MMAD) of 1-4 microns.[1] If more than one nebulizer is used in the testing of a given subject, nebulizer output should be measured for each nebulizer to assure a consistent dose. If output measurement is not possible, we recommend the use of the same nebulizer to deliver all concentrations to a given patient.

10.1.3 The gas powering the nebulizer and/or dosimeter should be at the correct driving pressure or flow (as specified by the manufacturer) and should be maintained at that pressure or flowrate consistently throughout the test.

10.1.4 Reagents:

10.1.4.1 The Food & Drug Administration (FDA) approved form of methacholine powder (Provocholine) is available in prepackaged vials ready for dilution. Provocholine and diluent can be obtained from Methapharm Inc, 131 Clarence St, Brantford, Ontario, Canada, N3T 2V6; Telephone 800.287.7686.

10.1.4.2 The recommended diluent used to dissolve the methacholine is sterile normal saline (0.9% sodium chloride) with or without a preservative (eg, 0.4% phenol).[2]

10.1.4.3 Various strategies have been described for dosing schemes.[10,13,34-38] The range of doses is 0.02-25.0 mg/mL, generally given in doubling doses[1,10,13,24,29] (ie, 0.02 mg/mL, 0.04 mg/mL, 0.08 mg/mL). The dosing scheme most recently recommended by the ATS is: diluent, 0.03, 0.06, 0.125, 0.25, 0.5, 1, 2, 4, 8, and 16 mg/mL.[2] If a shortened dosing protocol is desired, the ATS recommends: diluent, 0.06, 0.25, 1, 4, and 16 mg/mL.[2] Caution should be used with the shortened protocol when testing small children with asthma symptoms. The use of the diluent step is optional.[2]

10.1.4.4 In general, higher concentrations of methacholine solution (ie, > 1.25 mg/mL) are stable for at least 4 months when stored at 4° C.[39-41] The

package insert for Provocholine recommends that solutions > 0.25 mg/mL be stored for no longer than 2 weeks, with weaker solutions mixed on the day of testing.[13]

10.1.4.5 A pharmacist or other well-trained individual should prepare the methacholine reagents according to the manufacturer's recommendations, using sterile technique.

10.1.4.6 Reagents should be clearly labeled with dose, date prepared, and expiration date.

10.1.4.7 The test should be administered in a well-ventilated room (with at least 2 complete air exchanges per hour).[28] A filter to collect excess particles or an exhaust system to remove provocative material from the room may be desirable.

10.1.4.8 Oxygen, bronchodilators, and resuscitation equipment should be readily available.[1,24]

10.1.4.9 The need for written consent should be determined within the specific institution.

10.1.4.10 A pretest questionnaire should be used. An example of a questionnaire can be found in the ATS Methacholine Challenge Guideline.[2]

10.2 Personnel:

10.2.1 Methacholine challenge tests should be performed under the direction of a physician trained in pulmonary function testing and experienced in bronchial provocation. Personnel performing the test should be experienced in patient assessment, knowledgeable of and have demonstrated competency in performing this challenge (including reversal of methacholine response), know the associated hazards, and be certified in basic life support. Attainment of the CPFT and/or RPFT credentials is recommended.

10.2.2 During the testing procedure, a physician knowledgeable in provocation testing procedures and trained to treat acute bronchospasm and use resuscitation equipment must be close enough to respond in an emergency.

MCT 11.0 PATIENT MONITORING:

11.1 The FEV_1 is the primary variable to be monitored, and the results of spirometry should meet acceptability and reproducibility recommendations proposed by the ATS.[28] A shortened expiratory maneuver can be used in some situations and may be acceptable, and reproducibility after inhalation of some methacholine concentrations may be difficult.[2]

11.2 The test should be administered according to the specific protocol, with the number of breaths and the breathing pattern documented.

11.3 Breath sounds, pulse rate, pulse oximetry, and/or blood pressure may be monitored to assist in patient evaluation and test interpretation.[42-45] Patients should not be left unattended during the procedure.

11.4 In the case of a positive response to provocation (ie, $\geq$ 20% fall in FEV_1), bronchodilator may be administered to speed recovery. Spirometry should be repeated after bronchodilator administration to ensure that ventilatory function has returned to near baseline (ie, at least 85% of baseline).[46]

MCT 12.0 FREQUENCY:

12.1 To ensure that a previous methacholine challenge test does not affect a later test, 230 minutes should be allowed to elapse before the test is repeated.[47] Tolerance of methacholine may occur in patients who are not asthmatic when tests are repeated at less than 24-hour intervals.[48,49]

12.2 When a test is to be repeated, medications, exposures, time of day, and nebulizer employed should be held constant, if possible.

MCT 13.0 INFECTION CONTROL:

13.1 The staff, supervisors, and physician-directors associated with the pulmonary laboratory should be conversant with "Guideline for Isolation Precautions in Hospitals"[50] and develop and implement policies and procedures for the laboratory that comply with its recommendations for Standard Precautions and Transmission-Based Precautions.

13.2 The laboratory's manager and its medical director should maintain communication and cooperation with the institution's infection control service and the personnel health service to help assure consistency and thoroughness in

complying with the institution's policies related to immunizations, post-exposure prophylaxis, and job- and community-related illnesses and exposures.[51]

13.3 Primary considerations include adequate handwashing,[52] provision of prescribed ventilation with adequate air exchanges,[53] careful handling and thorough cleaning and processing of equipment,[50] and the exercise of particular care in scheduling and interfacing with the patient in whom a diagnosis has not been established.[50]

13.4 Sterility of reagents should be maintained by proper storage and aseptic handling.

MCT 14.0 AGE-SPECIFIC ISSUES:

Test instructions and techniques should be given in a manner that takes into consideration the learning ability and communication skills of the patient being tested.

14.1 Neonatal: This CPG does not apply to neonatal populations.

14.2 Pediatric: This CPG is appropriate for children who can perform good quality spirometry or body plethysmography ≥ 5 years of age).

14.3 Geriatric: This CPG is appropriate for the geriatric population.

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REFERENCES

1. Sterk PJ, Fabbri LM, Quanjer PH, Cockcroft DW, O'Byrne PM, Anderson SD, et al. Airway responsiveness: standardized challenge testing with pharmacological, physical and sensitizing stimuli in adults. Report Working Party Standardization of Lung Function Tests. European Community for Steel and Coal. Official Statement of the European Respiratory Society. Eur Respir J Suppl 1993 Mar;16:53-83.

2. Crapo RO, Casaburi R, Coates AL, Enright PL, Hankinson JL, Irvin CG, et al. Guidelines for methacholine and exercise challenge testing, 1999. This official statement of the American Thoracic Society was adopted by the ATS Board of Directors, July 1999. Am J Respir Crit Care Med 2000;161(1):309-329.

3. Chatham M, Bleeker ER, Smith PL, Rosenthal RR, Mason P, Norman PS. A comparison of histamine, methacholine, and exercise airway reactivity in normal and asthmatic subjects. Am Rev Respir Dis 1982;126(2):235-240.

4. Greenspon LW, Gracely E. A discriminate analysis applied to methacholine bronchoprovocation testing improves classification of patients as normal, asthma, or COPD. Chest 1992;102(5):1419-1425.

5. Cockcroft DW, Hargreave FE. Airway hyperresponsiveness: relevance of random population data to clinical usefulness. Am Rev Respir Dis 1990;142(3):497-500.

6. Cockcroft DW, Murdock KY, Berscheid BA, Gore BP. Sensitivity and specificity of histamine PC_{20} determination in a random selection of young college students. J Allergy Clin Immunol 1992;89(1 Pt 1):23-30.

7. Backer V, Groth S, Dirksen A, Bach-Mortensen N, Hansen KK, Laursen EM, Wendelboe D. Sensitivity and specificity of the histamine challenge test for the diagnosis of asthma in an unselected sample of children and adolescents. Eur Respir J 1991;4(9):1093-1100.

8. Perpina M, Pellicer C, de Diego A, Compte L, Macian V. Diagnostic value of the bronchial provocation test with methacholine in asthma: a Bayesian analysis approach. Chest 1993;104(1):149-154.

9. Fish JE. Bronchial challenge testing. In: Middleton E, editor. Allergy: principles and practice. 4th ed. St Louis: Mosby Year Book; 1993.

10. Chai H, Farr RS, Froehlich LA, Mathison DA, McLean JA, Rosenthal RR, et al. Standardization of bronchial inhalation challenge procedures. J Allergy Clin Immunol 1975;56(4):323-327.

11. Empey DW, Laitinen LA, Jacobs L, Gold WM, Nadel JA. Mechanisms of bronchial hyperreactivity in normal subjects after upper respiratory tract infection. Am Rev Respir Dis 1976;113(2):131-139.

12. Cheung D, Dick EC, Timmers MC, de Klerk EP, Spaan WJ, Sterk PJ. Rhinovirus inhalation causes long-lasting excessive airway narrowing in response to methacholine in asthmatic subjects in vivo. Am J Respir Crit Care Med

1995;152(5 Pt 1):1490-1496.

13. Package insert. Provocholine. Methapharm Inc. 131 Clarence St, Brantford, Ontario, Canada, N3T 2V6. Telephone 800.287.7686.

14. O'Connor BJ, Towse LJ, Barnes PJ. Prolonged effect of tiotropium bromide on methacholine-induced bronchoconstriction in asthma. Am J Respir Crit Care Med 1996;154(4 Pt 1):876-880.

15. Juniper EF, Kline PA, Vanzieleghem MA, Ramsdale EH, O'Byrne PM, Hargreave FE. Effect of long-term treatment with an inhaled corticosteroid (budesonide) on airway hyperresponsiveness and clinical asthma in nonsteroid-dependent asthmatics. Am Rev Respir Dis 1990;142(4):832-836.

16. Freezer NJ, Croasdell H, Doull IJM, Holgate ST. Effect of regular inhaled beclomethasone on exercise and methacholine airway responses in school children with recurrent wheeze. Eur Respir J 1995;8(9):1488-1493.

17. Jensen EJ, Dahl R, Steffensen F. Bronchial reactivity to cigarette smoke in smokers: repeatability, relationship to methacholine reactivity, smoking and atopy. Eur Respir J 1998;11(3):670-676.

18. Lemiere C, Cartier A, Dolovich J, Chan-Yeung M, Grammer L, Ghezzo H, et al. Outcome of specific bronchial responsiveness to occupational agents after removal from exposure. Am J Respir Crit Care Med 1996;154(2 Pt 1):329-333.

19. Cockcroft DW, Swystun VA, Bhagat R. Interaction of inhaled beta-2 agonist and inhaled corticosteroid on airway responsiveness to allergen and methacholine. Am J Respir Crit Care Med 1995;152(5 Pt 1):1485-1489.

20. Anderson SD. Exercise-induced asthma: the state of the art. Chest 1975;87 (Suppl 5):S191-S197.

21. Godfrey S, Silverman M, Anderson SD. Problems of interpreting exercise-induced asthma. J Allergy Clin Immunol 1973;52(4):199-209.

22. Edmunds AT, Tooley M, Godfrey S. The refractory period after exercise-induced asthma: its duration and relation to the severity of exercise. Am Rev Respir Dis 1978;117(2):247-254.

23. American Association for Respiratory Care. AARC Clinical Practice Guideline: Spirometry, 1996 update. Respir Care 1996;41(7):629-636.

24. Spector SL. Bronchial inhalation challenges with aerosolized bronchoconstrictive substances. In: Spector SL, editor. Provocative challenge procedures. Boca Raton FL: CRC Press; 1983: 148.

25. Orehek J, Nicoli MM, Delpierre S, Beaupre A. Influence of the previous deep inspiration on the spirometric measurement of provoked bronchoconstriction in asthma. Am Rev Respir Dis 1981;123(3):269-272.

26. Gayrard P, Orehek J, Grimaud C, Charpin J. Bronchoconstrictor effects of a deep inspiration in patients with asthma. Am Rev Respir Dis 1975;111(4):433-439.

27. Lloyd TC. Bronchoconstriction in man following single deep inspirations. J Appl Physiol 1963;18:114-116.

28. American Thoracic Society. Standardization of spirometry, 1994 update. Am J Respir Crit Care Med 1995;152(3):1107-1136.

29. Merkus PJFM, van Essen-Zandvliet EEM, Parlevliet E, Borsboom G, Sterk PJ, Kerrebijn KF, Quanjer PH. Changes of nebulizer output over the years. Eur Respir J 1992;5(4):488-491.

30. Ryan G, Dolovich MB, Obminski G, Cockcroft DW, Juniper E, Hargreave FE, Newhouse MT. Standardization of inhalation provocation tests: influence of nebulizer output, particle size, and method of inhalation. J Allergy Clin Immunol 1981;67(2):156-161.

31. National Committee for Clinical Laboratory Standards (NCCLS). GP26-A A quality system model for health care: approved guideline (1999). Available from NCCLS: phone 610-688-0100; Fax 610-688-0700;e-mail exoffice@ nccls.org.

32. National Committee for Clinical Laboratory Standards (NCCLS). NCCLS. HS4-A A quality system model for respiratory care: approved guideline. Available from NCCLS: phone 610-688-0100; Fax 610-688-0700; e-mail exoffice@nccls.org.

33. American Association for Respiratory Care. AARC Clinical Practice Guideline: Body plethysmography. 2001 revision & update. Respir Care 2001;46(5):506-513.

34. Chatham M, Bleecker ER, Norman P, Smith PL, Mason P. A screening test for airways reactivity: an abbreviated methacholine inhalation challenge. Chest 1982;82(1):15-18.

35. Schmidt LE, Thorne PS, Watt JL, Schwartz DA. Is an abbreviated bronchial challenge with histamine valid? Chest 1992;101(1):141-145.

36. Yan K, Salome C, Woolcock AJ. Rapid method for measurement of bronchial responsiveness. Thorax 1983;38(10):760-765.

37. Corrao WM, Braman SS, Irwin RS. Chronic cough as the sole presenting manifestation of bronchial asthma. N Engl J Med 1979;300(12):633-637.

38. Cockcroft DW, Killian DN, Mellon JJ, Hargreave FE. Bronchial reactivity to inhaled histamine: a method and clinical survey. Clin Allergy 1977;7(3):235-243.

39. MacDonald NC, Whitmore CK, Makoid MC, Cobby J. Sta

bility of methacholine chloride in bronchial provocation test solutions. Am J Hosp Pharm 1981;38(6):868-871.

40. Pratter MR, Woodman TF, Irwin RS, Johnson B. Stability of stored methacholine chloride solutions: clinically useful information. Am Rev Respir Dis 1982;126(4):717-719.

41. Hayes RD, Beach JR, Rutherford DM, Sim MR. Stability of methacholine chloride solutions under different storage conditions over a 9-month period. Eur Respir J 1998;11(4):946-948.

42. Avital A, Bar-Yishay E, Springer C, Godfrey S. Bronchial provocation tests in young children using tracheal auscultation. J Pediatr 1988;112(4):591-594.

43. Wilson NM, Phagoo SB, Silverman M. Use of transcutaneous oxygen tension, arterial oxygen saturation, and respiratory resistance to assess the response to inhaled methacholine in asthmatic children and normal adults. Thorax 1991;46(6):433-437.

44. Sprikkelman AB, Grol MH, Lourens MS, Gerritsen J, Heymans HS, van Aalderen WM. Use of tracheal auscultation for the assessment of bronchial responsiveness in asthmatic children. Thorax 1996;51(3):317-319.

45. Phagoo SB, Wilson NM, Silverman M. Repeatability of methacholine challenge in asthmatic children measured by change in transcutaneous oxygen tension. Thorax 1992;47(10):804-808.

46. Tashkin DP, Altose MD, Bleecker ER, Connett JE, Kanner RE, Lee WW, Wise R. The lung health study: airway responsiveness to inhaled methacholine in smokers with mild to moderate airflow limitation. The Lung Health Study Research Group. Am Rev Respir Dis 1992;145(2 Pt 1):301-310.

47. Cartier A, Malo JL, Begin P, Sestier M, Martin RR. Time course of the bronchoconstriction induced by inhaled histamine and methacholine. J Appl Physiol 1983;54(3):821-826.

48. Stevens WHP, Manning PJ, Watson RM, O'Byrne PM. Tachyphylaxis to inhaled methacholine in normal but not asthmatic subjects. J Appl Physiol 1990;69(3):875-879.

49. Beckett WS, Marenberg ME, Pace PE. Repeated methacholine challenge produces tolerance in normal but not in asthmatic subjects. Chest 1992;102(3):775-779.

50. Garner JS. Guideline for isolation precautions in hospitals. Part 1. Evolution of isolation practices. Hospital Infection Control Practices Advisory Committee. Atlanta GA: Centers for Disease Control and Prevention, 1-01-1996. www.cdc.gov or Am J Infect Control 1996;24(1):24-31.

51. Centers for Disease Control and Prevention. Hospital Infection Control Practices Advisory Committee. Guideline for infection control in healthcare personnel, 1998. Am J Infect Control 1998;26:269-354 or Infect Control Hosp Epidemiol 1998;19(6):407-463.

52. Larson EL. APIC guideline for handwashing and hand antisepsis in health care settings. Am J Infect Control 1995;23(4):251-269.

53. Centers for Disease Control. Guidelines for preventing the transmission of Mycobacterium tuberculosis in health-care facilities 1994. MMWR 1994;43(RR-13):1-132. Federal Register 1994;59(208):54242-54303.

AARC Clinical Practice Guideline

Static Lung Volumes: 2001 Revision & Update

SLV 1.0 PROCEDURE:

Measurement of static lung volumes and capacities in adults and in children (age $\geq$ 5). This guideline focuses on commonly used techniques for measuring lung volumes, including spirometry, gas-dilution determination of functional residual capacity (FRC), and whole-body plethysmography determination of thoracic gas volume (VTG). Other methods (eg, single-breath nitrogen, single-breath helium, and roentgenologic determinations of lung volumes) are not discussed in this document, but may be useful in certain situations.

SLV 2.0 DESCRIPTION/DEFINITIONS:

2.1 Static lung volumes are determined using methods in which airflow velocity does not play a role. The sum of two or more lung-volume subdivisions constitutes a lung capacity. The subdivisions and capacities are expressed in liters at body temperature and pressure saturated with water vapor (BTPS).
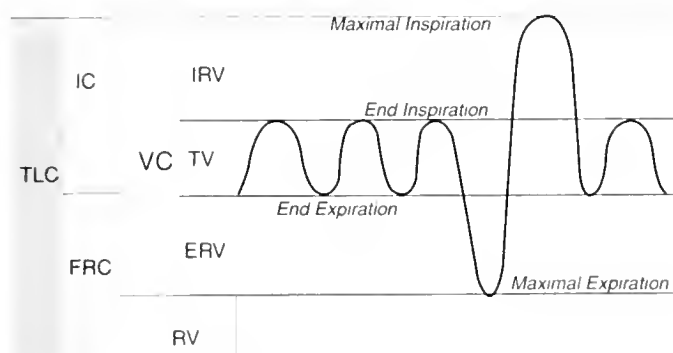
Fig. 1. Subdivisions of Lung Volume

2.2 Tidal volume is the volume of air that is inhaled or exhaled with each respiratory cycle.[1] (Although both V_T and TV have been used to denote this volume, TV is used in this guideline.) It varies with the conditions under which it is measured (eg, rest, exercise, posture).

When TV is reported, an average of at least 6 breaths should be used.[2] (Fig. 1)

2.3 Inspiratory reserve volume (IRV) is the maximal volume of air that can be inhaled from TV end-inspiratory level.[2]

2.4 Expiratory reserve volume (ERV) is the maximal volume of air that can be exhaled after a normal tidal exhalation (ie, from functional residual capacity, or FRC).[2]

2.5 Residual volume (RV) is the volume of gas remaining in the lung at the end of a maximal expiration.[1] It may be calculated by subtracting ERV from FRC (RV = FRC – ERV) or by subtracting vital capacity (VC) from total lung capacity, or TLC (RV = TLC – VC).

2.6 Inspiratory capacity (IC) is the maximal volume of air that can be inhaled from the tidal-volume end-expiratory level (ie, FRC). It is equal to the sum of TV and IRV.[2]

2.7 Vital capacity (VC) is the volume change that occurs between maximal inspiration and maximal expiration. The subdivisions of the VC include TV, inspiratory reserve volume (IRV), and expiratory reserve volume (ERV). The largest of three technically satisfactory VC maneuvers should be reported. The two largest VCs should agree within 5% or 100 mL, whichever is larger. The volume change can be accomplished in several ways.[2]

2.7.1 Two-stage VC: a slow maximal inspiration from TV end-expiratory level after a normal exhaled TV, followed by quiet breathing, followed by a slow maximal expiration from TV (ie, end-expiratory level, or functional residual capacity (ie, FRC). The reverse maneuver is also acceptable:

2.7.2 Forced vital capacity (FVC): the volume of air exhaled during a forced maximal expiration following a forced maximal inspiration. The FIVC is the forced VC obtained during a maximal inspiration following a maximal expiration.

2.8 FRC is the volume of air in the lung at the average TV end-expiratory level. It is the sum of the ERV and RV. When subdivisions of lung volume are reported, the method of measurement should be specified (eg, helium dilution, nitrogen washout, body plethysmography).[2]

2.9 Thoracic gas volume (VTG) is the volume of air in the thorax at any point in time and at any level of thoracic expansion. It is usually measured by whole-body plethysmography. It may be determined at any level of lung inflation; however, it is most commonly determined at or near FRC.[2] As an alternative, lung volume may be tracked continuously, and FRC determined from VTG by addition or subtraction of volume.

2.10 Total lung capacity (TLC) is the volume of air in the lung at the end of a maximal inspiration. It is usually calculated in one of two ways: (1) TLC = RV + VC or (2) TLC = FRC + IC. The method of measurement (eg, gas dilution, body plethysmography) should be specified.[2]

SLV 3.0 SETTINGS:

3.1 Pulmonary function laboratories

3.2 Cardiopulmonary laboratories

3.3 Clinics and physicians' offices

3.4 Patient care areas

3.5 Study and field settings

SLV 4.0 INDICATIONS:

Indications include but are not limited to the need

4.1 to diagnose restrictive disease patterns;[3]

4.2 to differentiate between obstructive and restrictive disease patterns,[2] particularly in the presence of a reduced VC;[4]

4.3 to assess response to therapeutic interventions (eg, drugs, transplantation, radiation, chemotherapy, lobectomy, lung-volume-reduction surgery);

4. 4 to aid in the interpretation of other lung function tests (eg, DL/VA, sG$_{aw}$, RV/TLC;[2]

4. 5 to make preoperative assessments[2] in patients with compromised lung function (known or suspected) when the surgical procedure is known to affect lung function;

4. 6 to provide an index of gas trapping (by comparison of gas dilution techniques with plethysmographic measurements).[5]

SLV 5.0 CONTRAINDICATIONS:

5.1 No apparent absolute contraindications exist; the relative contraindications for spirometry are appropriate and may include:[2,6,7]

5.1.1 hemoptysis of unknown origin;

5.1.2 untreated pneumothorax;

5.1.3 pneumothorax treated with a chest tube—because the chest tube may introduce leaks and interfere with gas-dilution measurements;

5.1.4 unstable cardiovascular status;

5.1.5 thoracic and abdominal or cerebral aneurysms.

5.2 With respect to whole-body plethysmography, such factors as claustrophobia, upper body paralysis, obtrusive body casts, intravenous (I.V.) pumps, or other conditions that immobilize or prevent the patient from fitting into or gaining access to the 'body box' are a concern. In addition, the procedure may necessitate stopping I.V. therapy or supplemental oxygen.

SLV 6.0 HAZARDS/COMPLICATIONS:

6.1 Infection may be contracted from improperly cleaned tubing, mouthpieces, manifolds, valves, and pneumotachometers.

6.2 Hypoxemia may result from interruption of O_2 therapy in the body box.

6.3 Ventilatory drive may be depressed in susceptible subjects (ie, some CO_2 retainers) as a consequence of breathing 100% oxygen during the nitrogen washout.[8] Such patients should be carefully observed.

6.4 Hypercapnia and/or hypoxemia may occur during helium-dilution FRC determinations as a consequence of failure to adequately remove CO_2 or add O_2 to the rebreathed gas.

SLV 7.0 LIMITATIONS OF METHODOLOGY/ VALIDATION OF RESULTS:

7.1 Patient-related limitations:

7.1.1 Slow VC is effort-dependent and requires understanding and motivation on the subject's part. Physical and/or mental impairment may limit patient's ability to perform.

7.1.2 Some patients may be unable to perform the necessary panting maneuver required for plethysmographic determination of FRC.

7.1.3 Some subjects are unable to maintain mouth seal or cooperate adequately for the

time necessary to perform the test. Cough is a common cause of such limitations.

7.1.4 Certain pathologic conditions in the subject can cause a leak in a lung-volume-measurement system (eg, perforated eardrum, tracheostomy, transtracheal catheter, chest tube).

7.1.5 FRC measured by gas dilution may be underestimated in individuals with airflow limitation and air trapping.[9,10] Body plethysmography may overestimate FRC in subjects with severe airway obstruction or induced bronchospasm at panting frequencies greater than 1 Hz (1 cycle/second).[11-13]

7.1.6 Elimination of nitrogen from tissues and blood can result in overestimation of the FRC in healthy subjects unless appropriate corrections are made.[2]

7.2 Test validation encompasses those calibration and procedural elements that help assure credible results:

7.2.1 Spirometry

7.2.1.1 Spirometers (volume-displacement devices or flow-sensing devices) should meet the American (1994) and European Thoracic Societies' (1993) current accepted standards.[2,3] Volume-displacement spirometers should be leak tested when calibrated (eg, daily).[14]

7.2.1.2 The VC should be measured as close as possible in time to the FRC determination.[2]

7.2.2 Gas-dilution methods for FRC determination:

7.2.2.1 Open-circuit multibreath nitrogen washout method

7.2.2.1.1 Test should be continued for 7 minutes or until N_2 concentration falls below 1.0%.[15] In subjects with airflow obstruction and air trapping, the time period for measuring FRC may need to be extended.

7.2.2.1.2 A minimum of 15 minutes should elapse before test is repeated.[16]

7.2.2.1.3 Initial alveolar nitrogen concentration of 80% can be assumed[2] if patient has been breathing room air for at least 15 minutes.

7.2.2.2 Closed-circuit multibreath helium equilibration method

7.2.2.2.1 The helium concentration should be measured at least every 15 seconds, and water vapor should be removed from the fraction of gas that is introduced into the helium analyzer.[2] The reference cell of the He katharometer should also have a water absorber in-line, if room air is used for zeroing.

7.2.2.2.2 A mixing fan should circulate and completely mix the air throughout the main circuit.

7.2.2.2.3 The breathing valve and mouthpiece (without a filter) should add < 60 mL dead space to the system for adults and a proportionately reduced increase for pediatric subjects and should be easy to disassemble for cleaning.

7.2.2.2.4 Gas mixing is considered complete when the change in helium concentration has been constant over a 2-minute period (ie, changes less than 0.02%) or 10 minutes has elapsed.[4] If the helium concentration can be read directly or processed by computer, helium equilibration can be assumed when the change is < 0.02% in 30 seconds.[2]

7.2.2.2.5 The need to correct for body absorption of helium is controversial.

7.2.2.2.6 The delay between the repeated measurements should be at least the same as the time taken to reach equilibrium or 5 minutes, whichever is greater.[17,18]

7.2.4 Whole body plethysmography

7.2.4.1 The frequency of panting breathing movements against the shutter should be 1 cycle/second.[11,13,19]

7.2.4.2 The cheeks and chin should be firmly supported with both hands. This should be done without supporting the elbows or elevating the shoulders.[20]

7.2.4.3 Plethysmographic determination of FRC is the method of choice in patients with airflow limitation and air trapping.[2]

7.2.4.4 This method may be the more practical method in subjects with short attention spans or inability to stay on the mouthpiece (eg, children).

7.3 Reproducibility of results is essential to validation and test quality.

7.3.1 Multiple FRC determinations by gas dilution should be made, with at least two trials agreeing within 10% of the mean.[21]

7.3.2 FRC determinations by body plethysmography (at least 3 separate trials) should agree within 5% of the mean.[22]

7.3.3 IC and ERV measurements should agree within 5% or 60 mL (of the mean) whichever is larger. In patients who have large variability, this should be noted.

7.3.4 The two largest VC measurements should agree within 200 mL.[3]

7.4 Clear and complete reporting of results is essential to test quality.

7.4.1 The average FRC value should always be reported (and should ideally include the variability).

7.4.2 The largest volume of either VC or FVC should be reported

7.4.3 The largest reproducible value should be reported for IC and ERV, as described in 7.3.3.

7.4.4 Various methods are used for calculating TLC and RV.[23] The consensus of the Committee is that the two acceptable methods for reporting TLC and RV from FRC determinations made using gas dilution techniques are:

TLC = mean FRC + largest IC,
RV = TLC − largest VC; or
RV = mean FRC − largest ERV,
TLC = RV + largest VC.

For body plethysmographic determinations, a VC maneuver (with its IC and ERV subdivisions) should be performed in conjunction with each VTG maneuver and the TLC calculated as

TLC = FRC + IC.*

*(Note: the mean IC should be close to the largest IC)

The reported TLC should be the mean of all acceptable maneuvers; the RV should be calculated as:

RV = mean TLC − largest VC.

7.5 Conditions under which testing is done can affect results and should be controlled to the extent possible. If certain conditions cannot be met, the written report should reflect that.

7.5.1 Lung volumes are influenced by body position[24,25] and should be made in the sitting position. If another position is used, it should be noted.[2]

7.5.2 Breathing movements should not be restricted by clothing.

7.5.3 Diurnal variations in lung function may cause differences and, thus, if serial measurements are to be performed, the time of the day that measurements are made should be held constant.[2]

7.5.4 The patient should not have smoked for at least 1 hour prior to the measurements.

7.5.5 The patient should not have had a large meal shortly before testing.

7.5.6 Nose clips should always be worn during testing.[2]

7.5.7 Measurements made at ambient temperature and pressure saturated with water vapor (ATPS) conditions are corrected to body temperature and pressure saturated with water vapor (BTPS) conditions.

7.5.8 No corrections are necessary for altitude because no consistent differences in lung volumes (TLC, VC, FRC, and RV) due solely to altitude have been found from sea level up to 1,800 meters.[26-28]

7.5.9 After the mouthpiece is in place, the patient should be asked to breathe quietly in order to become accustomed to the apparatus and attain a stable breathing pattern. The end-expiratory level should be reproducible within 100 mL.

7.5.10 VC can be measured before disconnecting the patient from measuring systems. As an alternative, the patient can be disconnected and the VC performed immediately afterward.

7.5.11 If expired VC is measured with a CO_2 absorber in the system, an appropriate volume correction must be made. ($1.05 \times$ expired volume is the correction commonly incorporated into commercial software.)

7.5.12 If a filter is used during FRC measurement, the filter volume must be subtracted.

7.6 Choice of reference values may affect interpretation.

7.6.1 Make a tentative selection from published reference values. The characteristics of the healthy reference population should match the study group with respect to age, body size, gender, and race. The equipment, techniques, and measurement conditions should be similar.

7.6.2 Following selection of apparently appropriate reference values, compare measurements obtained from a representative sample of healthy individuals (10-20 subjects) over an appropriate age range to the predicted values obtained from the selected reference values. If an appreciable number of the sample falls outside of the normal range, more appropriate reference values should be sought. This procedure detects only relatively gross differences between sample and reference population.[29]

7.6.3 Predicted values for RV, FRC, and TLC should be derived from the same reference population.

7.7 Expression of results

7.7.1 The upper and lower limits of normal may be derived from the standard error of the estimates (SEE) around the regression lines. The two-tail 95% confidence interval can be estimated by multiplying $\pm 1.96 \times$ SEE. A one-tailed 95% confidence interval can also be used for parameters in which only an abnormal high or low limit of normal is needed; the one-tailed limit is estimated by multiplying $\pm 1.64 \times$ SEE and subtracting this value from the mean.[22,24] These methods of estimating the limits of normal are applicable only if the reference data are normally distributed (Gaussian).[1]

7.7.2 The common practice of expressing results as percent predicted and regarding 80% predicted as the lower limit of normal is not valid unless the standard deviation (SD) of the reference data is proportional to the mean value.[30]

SLV 8.0 ASSESSMENT OF NEED (See SLV 4.0 Indications.)

Technologist-driven protocols (TDP) may be useful for assessing the need for lung-volume determination, particularly in the context of other pulmonary function results (eg, spirometry, diffusing capacity).
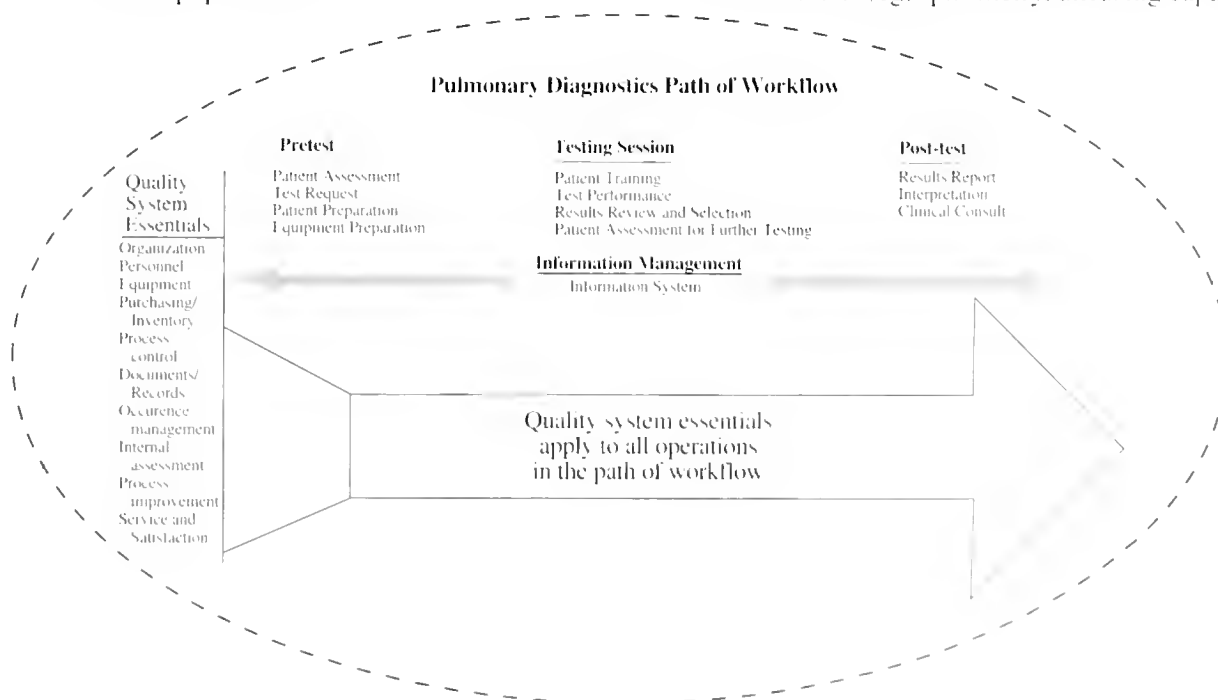
Fig. 2. Structure for a Quality System Model for a Pulmonary Diagnostics Service (From Reference 31, with permission)

SLV 9.0 ASSESSMENT OF QUALITY OF TEST AND VALIDITY OF RESULTS:

The consensus of the committee is that all diagnostic procedures should follow the quality model described in the NCCLS GP26-A A Quality System Model for Health Care.[31] (Fig. 2) The document describes a laboratory path of workflow model that incorporates all the steps of the procedure. This process begins with patient assessment and the generation of a clinical indication for testing through the application of the test results to patient care. The quality system essentials defined for all health care services provide the framework for managing the path of workflow. A continuation of this model for respiratory care services is further described in NCCLS HS4-A A Quality System Model for Respiratory Care.[32] In both quality models the patient is the central focus.

9.1 General considerations include:

9.1.1 As part of any quality assurance program, indicators must be developed to monitor areas addressed in the path of workflow.

9.1.2 Each laboratory should standardize procedures and demonstrate intertechnologist reliability. Test results can be considered valid only if they are derived according to and conform to established laboratory quality control, quality assurance, and monitoring protocols.

9.1.3 Documentation of results, therapeutic intervention (or lack of) and/or clinical decisions based on the testing should be placed in the patient's medical record.

9.1.4 The type of medications, dose, and time taken prior to testing and the results of the pretest assessment should be documented.

9.1.5 Report of test results should contain a statement by the technician performing the test regarding test quality (including patient understanding of directions and effort expended) and, if appropriate, which recommendations were not met.[2,3,33]

9.1.6 Test results should be interpreted by a physician, taking into consideration the clinical question to be answered.

9.1.7 Personnel who do not meet annual competency requirements or whose competency is deemed unacceptable as documented in an occurrence report should not be allowed to participate, until they have received remedial instruction and have been re-evaluated.

9.1.8 There must be evidence of active review of quality control, proficiency testing, and physician alert, or 'panic' values, on a level commensurate with the number of tests performed.

9.2 Calibration measures specific to equipment used in measuring lung volumes include:

9.2.1 Spirometers and/or other volume transducers should be calibrated daily using a 3-L syringe or another more sophisticated device.[3] Volume-based spirometers should be checked for leaks.

9.2.2 Gas dilution systems should have their gas analyzers, (ie, He, N_2, O_2, CO_2) calibrated according to the manufacturer's recommendations immediately before each test. Some analyzers may require more frequent calibration.

9.2.3 Gas conditioning devices such as CO_2 and water absorbers should be inspected daily.

9.2.4 Body plethysmographs (including each transducer) should be calibrated at least daily, according to the manufacturer's recommendations. Leak checks or calculation of time constants should be performed in accordance with the manufacturer's recommendations.

9.3 Quality control measures specific to measuring lung volumes include:

9.3.1 Lung volume analogs provide a means of checking the absolute accuracy and assessing precision. A 3-L syringe with/without an additional volume container can be used to check gas dilution systems (both open and closed circuit systems). As an alternative, a large-volume syringe can be used to assess the linearity of the associated gas analyzers, using a serial dilution technique.[34]

9.3.2 Isothermal bottles can be constructed

or purchased in order to check body plethysmograph function (volume accuracy).

9.3.3 Biologic controls should be used to assess the performance of the entire lung-volume system (transducers, gas analyzers, software). The means and standard deviations of 8-10 measurements of 2 or more healthy subjects may be used to check the precision of the system, as well as to troubleshoot when problems are suspected.

SLV 10.0 RESOURCES:

10.1 Equipment: Specifications should conform to recognized standards.

10.1.1 All spirometers (volumetric or flow-based) should meet or exceed the minimum recommendations of the American Thoracic Society.[3]

10.1.2 Helium analyzers (katharometers) should be linear from 0 to 10% with a resolution less than 0.05% He and an accuracy of 0.1%. The gas flow through the meter should be constant at 20 mL/min or more. The 95% response time of the system (analyzer, spirometer with fan) for a 2% step change should be ≤ 15 seconds.[2]

10.1.3 Plethysmographs should include:[2]

10.1.3.1 a patient compartment appropriate for the population to be tested;

10.1.3.2 a piston pump for box calibration and a manometer or similar device for mouth pressure calibration. A 3-liter syringe should be available for pneumotachometer calibration;

10.1.3.3 a vent to atmosphere (constant volume configurations);

10.1.3.4 a mouth shutter capable of closing within 0.1 seconds;

10.1.3.5 and an intercom for patient-technologist communication.

10.1.4 Nitrogen analyzers should have a range of 0-100% ± 0.5% with 50-millisecond response time or rapidly responding O_2 and CO_2 analyzers that allow calculation of the fraction of expired N_2 (FeN_2) should be incorporated.

10.2 Personnel

10.2.1 Lung-volume testing should be performed under the direction of a physi-cian trained in pulmonary diagnostics.

10.2.2 Personnel should be trained (with verifiable training and demonstrated competency) in all aspects of lung-volume determination, including equipment theory of operation, quality control, and test outcomes relative to diagnosis and/or medical history.[35]

10.2.3 Attainment of either the CPFT or RPFT credential is recommended by the Committee.

SLV 11.0 MONITORING:

The following should be monitored during lung-volume determinations:

11.1 reproducibility of repeated efforts;

11.2 presence or absence of adverse effects of testing on the patient during testing. (Patients on supplemental oxygen may require periods of time to rest on oxygen between trials.)

SLV 12.0 FREQUENCY:

The frequency of lung-volume measurements depends on the clinical status of the subject and the indications for performing the test.

SLV 13.0 INFECTION CONTROL:

13.1 The staff, supervisors, and physician-directors associated with the pulmonary laboratory should be conversant with "Guideline for Isolation Precautions in Hospitals"[36] and develop and implement policies and procedures for the laboratory that comply with its recommendations for Standard Precautions and Transmission-Based Precautions.

13.2 The laboratory's manager and its medical director should maintain communication and cooperation with the institution's infection control service and the personnel health service to help assure consistency and thoroughness in complying with the institution's policies related to immunizations, post-exposure prophylaxis, and job- and community-related illnesses and exposures.[37]

13.3 Primary considerations include adequate handwashing,[38] provision of prescribed ventilation with adequate air exchanges,[39] careful handling and thorough cleaning and processing of equipment,[36] and the exercise of particular care

in scheduling and interfacing with the patient in whom a diagnosis has not been established. Considerations specific for lung-volume measurement include:

13.3.1 The use of filters is neither recommended nor discouraged. Filters may be appropriate for use in systems that use valves or manifolds on which deposition of expired aerosol nuclei is likely.[40]

13.3.2 If filters are used in gas-dilution procedures, their volume should be subtracted when FRC is calculated.

13.3.3 If filters are used in the plethysmograph system, the resistance of the filters should be subtracted from the airways resistance calculation.

13.3.4 Nondisposable mouthpieces and equipment parts that come into contact with mucous membranes, saliva, and expirate should be cleaned and sterilized or subjected to high-level disinfection between patients.[36,41] Gloves should be worn when handling potentially contaminated equipment.

13.3.5 Flow sensors, valves, and tubing not in direct contact with the patient should be routinely disinfected according to the hospital's infection control policy. Any equipment surface that displays visible condensation from expired gas should be disinfected or sterilized before it is reused.

13.3.6 Water-sealed spirometers should be drained weekly and allowed to dry.[2]

13.3.7 Closed circuit spirometers, such as those used for He-dilution FRC determinations, should be flushed at least 5 times over their entire volume to facilitate clearance of droplet nuclei. Open circuit system need only have the portion of the circuit through which rebreathing occurs decontaminated between patients.

SLV 14.0 AGE-SPECIFIC ISSUES:

Test instructions should be provided and techniques described in a manner that takes into consideration the learning ability and communications skills of the patient being served.

14.1 Neonatal: This Guideline does not apply to the neonatal population.

14.2 Pediatric: These procedures are appropriate for children who can perform spirometry of acceptable quality and can adequately follow directions for plethysmographic testing.

14.3. Geriatric: These procedures are appropriate for members of the geriatric population who can perform spirometry of acceptable quality and adequately follow directions for plethysmographic testing.

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REFERENCES

1. ACCP-ATS Joint Committee on Pulmonary Nomenclature. Pulmonary terms and symbols. Chest 1975;67(5):583-593.
2. Quanjer PH, Tammeling GJ, Cotes JE, Pedersen OF, Peslin R, Yernault JC. Lung volumes and forced ventilatory flows. Report Working Party Standardization of Lung Function Tests. European Community for Steel and Coal. Official Statement of the European Respiratory Society. Eur Respir J Suppl 1993 Mar;16:5-40.
3. American Thoracic Society. Lung function testing: selection of reference values and interpretative strategies. Am Rev Respir Dis 1991;144:1202-1218.
4. Aaron SD, Dales RE, Cardinal P. How accurate is spirometry at predicting restrictive pulmonary impairment? Chest 1999;115: 869-873.
5. Wade JF, Mortenson R, Irvin CG. Physiologic evaluation of bullous emphysema. Chest 1991;100(4):1151-1154.
6. American Thoracic Society: Standardization of spirometry: 1994 update. Am J Respir Crit Care Med 1995;152(3):1107-1136.
7. American Association for Respiratory Care. AARC Clinical practice guideline: Spirometry, 1996 update. Respir Care 1996;41(7)629-636.
8. Miller WF, Scacci R, Gast LR. Laboratory evaluation of pulmonary function. Philadelphia: JB Lippincott, 1987: 137.
9. Bedell GN, Marshall R, DuBois AB, Comroe JH. Plethys-

mographic determination of the volume of gas trapped in the lungs. J Clin Invest 1956;35:664-670.

10. Ross JC, Copher DE, Teays JD, Lord JJ. Functional residual capacity in patients with pulmonary emphysema. Ann Intern Med 1962;57:18-28.

11. Rodenstein DO, Stanescu DC, Francis C. Demonstration of failure of body plethysmography in airway obstruction. J Appl Physiol 1982;52(4):949-954.

12. Shore SA, Huk O, Mannix S, Martin JG. Effect of panting frequency on the plethysmographic determination of thoracic gas volume in chronic obstructive pulmonary disease. Am Rev Respir Dis 1983;128(1):54-59.

13. Shore S, Milic-Emili J, Martin JG. Reassessment of body plethysmographic technique for the measurement of thoracic gas volume in asthmatics. Am Rev Respir Dis 1982;126(3):515-520.

14. Gardner RM, Crapo RO, Nelson SB. Spirometry and flow-volume curves. Clin Chest Med 1989;10(2):145-154.

15. Darling RC, Cournand A, Richards DW Jr. Studies on intrapulmonary mixture of gases. III. Open circuit methods for measuring residual air. J Clin Lab Invest 1940;19:609-618.

16. Clausen JL, editor. Pulmonary function testing: guidelines and controversies. New York: Academic Press, 1982.

17. British Thoracic Society and Association of Respiratory Technicians and Physiologists. Guidelines for the measurement of respiratory function. Respir Med 1994; 88:165-194.

18. Meneely GR, Ball CO, Kory RC, et al. A simplified closed circuit helium dilution method for the determination of the residual volume of the lungs. Am J Med 1960; 28:824-831.

19. Rodenstein DO, Stanescu DC. Frequency dependence of plethysmographic volume in healthy and asthmatic subjects. J Appl Physiol 1983;54(1):159-165.

20. American Thoracic Society. Wanger J, editor. Pulmonary function laboratory management and procedure manual. 1998:13-14.

21. Schanning CG, Gulsvik A. Accuracy and precision of helium dilution technique and body plethysmography in measuring lung volumes. Scand J Clin Lab Invest 1983;32:271-277.

22. DuBois AB, Botelho SY, Bedell GN, Marshal R, Comroe JH. A rapid plethysmographic method for measuring thoracic gas volume: a comparison with a nitrogen wash-out method for measuring functional residual capacity. J Clin Lab Invest 1956;35:322-326.

23. Bohadana AB, Teculescu D, Peslin R, Jansen da Silva JM, Pino J. Comparison of four methods for calculating the total lung capacity measured by body plethysmograph. Bull Eur Physiopathol Respir 1980; 16(6):769-776.

24. Burki NK. The effects of changes in functional residual capacity with posture on mouth occlusion pressure and ventilatory pattern. Am Rev Respir Dis 1977;116(5): 895-900.

25. Parot S, Chaudun E, Jacquemin E. The origin of postural variations of human lung volumes as explained by the effects of age. Respiration 1970;27(3):254-260.

26. Goldman HI, Becklake MR. Respiratory function tests: normal values at median altitude and the prediction of normal results. Am Rev Thorac Pulmon Dis 1969; 79:457-467.

27. Cotes JE, Saunders MJ, Adam JER, Anderson HR, Hall AM. Lung function in coastal and highland New Guineans - comparisons with Europeans. Thorax 1973;28(3):320-330.

28. Crapo RO, Morris AH, Clayton PD, Nixon CR. Lung volumes in healthy nonsmoking adults. Bull Eur Physiopathol Respir 1982;18(3):419-425.

29. Clausen JL. Prediction of normal values in pulmonary function testing. Clin Chest Med 1989;10(2):135-143.

30. Quanjer PH. Predicted values: how should we use them? (letter) Thorax 1988;43(8):663-664.

31. NCCLS. GP26-A. A quality system model for health care, approved guideline (1999). Available from NCCLS: phone 610-688-0100, Fax 610-688-0700, e-mail exoffice@ ncels.org.

32. NCCLS. HS4-A. A quality system model for respiratory care. Available from NCCLS: phone 610-688-0100, Fax 610-688-0700; e-mail exoffice@ncels.org.

33. Quanjer PH, Andersen LH, Tammeling GJ. Static lung volumes and capacities. Report of Working Party for Standardization of Lung Function Tests, European Community for Steel and Coal. Bull Eur Physiopathol Respir 1983;19(5, Suppl):11-21.

34. Ruppel GL. Manual of pulmonary function testing ,7th ed. St Louis: CV Mosby; 1998: 304-306.

35. Gardner RM, Clausen JL, Crapo RO, Epler GR, Hankinson JL, Johnson JL Jr, Plummer AL. American Thoracic Society Committee on Proficiency Standards for Clinical Pulmonary Laboratories. Quality assurance in pulmonary function laboratories. Am Rev Respir Dis 1986;134(3): 625-627.

36. Garner JS, Hospital Infection Control Practices Advisory, Centers for Disease Control and Prevention. Guideline for isolation precautions in hospitals. Am J Infect Control 1996;24(1):24-31 or http//www.apic.org/html/resc/gdisolat.html.

37. Centers for Disease Control and Prevention, Hospital Infection Control Practices Advisory Committee. Guideline for infection control in health care personnel, 1998. Am J Infect Control 1998;26:269-354 or Infect Control Hosp Epidemiol 1998;19(6):407-463.

38. Larson EL. APIC guideline for handwashing and hand antisepsis in health care settings. Am J Infect Control 1995;23(4):259-269.

39. Centers for Disease Control & Prevention. Guidelines for preventing the transmission of tuberculosis in health-care facilities, 1994. MMWR 1994;43(RR-13):1-32 or Federal Register 1994;59(208):54242-54303 or http://aepo-xdv-www.epo.cdc.gov/wonder/prevguid/m0035909/m0035909.htm

40. Kirk YL, Kenday K, Ashworth HA, Hunter PR. Laboratory evaluation of a filter for the control of cross-infection during pulmonary function testing. J Hosp Infect 1992;20:193-198.

41. Rutala WA. APIC guideline for selection and use of disinfectants. Am J Infect Control 1990;18(2):99-117.

call for abstracts

RESPIRATORY CARE • OPEN FORUM 2001

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the OPEN FORUM during the AARC International Respiratory Congress in San Antonio, Texas, December 1-4, 2001. Accepted abstracts will be published in the October 2001 issue of RESPIRATORY CARE. Membership in the AARC is not required for participation. All accepted abstracts are automatically considered for ARCF research grants.

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New Products & Services

Sleep Apnea Diagnosis. Sleep Solutions™ Inc. has received Food and Drug Administration marketing clearance for its Bedbugg™ At-Home Diagnostic System for evaluating snoring and other forms of upper airway obstruction in sleep. According to the company, this system allows remote evaluation of patient snoring and testing for obstructive sleep apnea. Press materials explain that the patient applies small sensors to his upper lip, chest, and fingertip and then only has to press the "start" button. Sleep Solutions says the system records data for up to three nights and that the company provides a comprehensive summary of the analysis to the physician either by fax or through a password-protected Web site. For more information from Sleep Solutions, circle number 154 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Cardiac Patient Simulator. Armstrong Medical offers *Rhythm*SIM™ Basic and Advanced Patient Simulators for practice in ACLS and PALS training. The company says the device allows students to learn cardiac rhythms in real time, offering more than 60 simulated rhythms. According to Armstrong, the *Rhythm*SIM™ can use a standard ECG monitor, simulates between 1 and 12 leads while operating on both AC and DC power, and is fully portable with up to 20 hours of battery power. Press materials also explain

the system offers optional television interface displaying rhythms on a standard set which is useful in teaching large groups. For more information from Armstrong Medical circle number 155 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Portable Blood Gas Analyzer. According to Radiometer™, their new ABL77 is the world's fastest portable blood gas analyzer providing results in less than one minute. The company describes the new device as created specifically for point-of-care testing with simple design making it very low maintenance and sophisticated control software providing precise results. Radiometer press materials say the ABL77 measures pH, P_{O_2}, P_{CO_2}, cCa^{2+}, cNa^+, cK^+, and Hct using just 85 µL of whole blood. For more information from Radiometer, circle number 156 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/
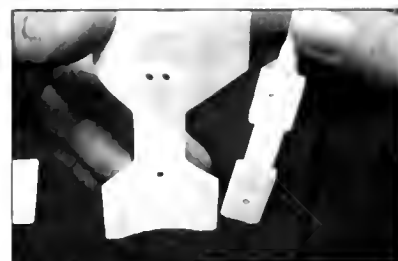
Pulse Oximetry Sensor. SIMS BCI Inc introduces their D.O.T. Sensors (digit oximeter transducer), ideal for use with all their pulse oximetry products. According to the company, their new sensors offer the convenience of a disposable with the durability of a reusable sensor to provide cost savings in patient care. SIMS BCI describes the sensors as uniquely designed to provide a better fit for a wider range of patient types and sizes; is completely latex free; and requires only one additional inventory item to support its use. For more information from SIMS BCI Inc, circle number 157 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Inservice Video. Passy-Muir announces its new clinical inservice video designed to provide clinicians with step-by-step instruction on the assessment, placement and transitioning issues related to placement of Passy-Muir Speaking Valves on both tracheostomized and ventilator dependent adult, pediatric, and neonatal patients. The company says the video is divided into four sections: Clinical Inservice; A Special Tribute to David Muir, Inventor; The Passy-Muir Tracheostomy and Ventilator Speaking Valves with Pediatrics; and Dysphagia Issues and Videoflouroscopy Clinical Lab. For more information from Passy-Muir, circle number 158 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Date	AARC & Affiliates Programs	Contact
May 2–4	Missouri Society for Respiratory Care's 30th Annual Meeting; **Lake of the Ozark, MO**	Diane Oldfather, (573) 458-0160, ext. 16194; dolfather@rolla.k12.mo.us, http://msrc.thehospital.com
May 9–10	Maine Society for Respiratory Care's Spring Fling; **Lewiston, ME**	Roberta Crockett, (207) 262-2214
May 11	ASRC Diamond Conference, **North Little Rock, AR**	Arkansas Children's Hospital, UA Medical Sciences, Kesha Mack, (501) 661-7962, mackkeshav@exchange.uams.edu
June 6–8	FSRC State Convention; **Fort Lauderdale, FL**	Pat Nolan, (561) 546-1863, (800) 447-3772, fsrc@inetw.net
June 13–15	New Jersey Society for Respiratory Care's 14th Annual NJ/NY Spring Forum, **Round Top, NY**	Ken Wyka, (201) 725-2528; or Bob Fluck, (315) 464-5580
June 13–15	Illinois Society for Respiratory Care's 33rd Annual Convention; **Oak Brook Terrace, IL**	Kelli DeBerry, (847) 981-3581, www.isrc.org
July 21–23	Management and Education Sections, Summer Forum; **Naples, FL**	AARC, (972) 243-2272, www.aarc.org
July 23–24	Asthma Disease Management Seminar, **Naples, FL**	AARC, (972) 243-2272, www.aarc.org
Sept. 12–14	Alabama Society for Respiratory Care's Annual Meeting, **Birmingham, AL**	Bill Pruitt, (334) 434-3405, wpruitt@jaguar1.usouthal.edu
Sept. 26–27	MSRC Annual Meeting, **Sturbridge, MA**	Valeri-Ann Bolduc, (508) 429-7478, O2val@aol.com
Dec. 1–4	47th International Respiratory Congress; **San Antonio, TX**	AARC, (972) 243-2272, www.aarc.org
Date	**Other Meetings**	**Contact**
May 12–14	Spring Sleep Seminar 2001, **Branson, MO**	Bill Rivers or Melinda Trimble, (501) 713-1272
Oct. 2–4	Cardiorespiratory Diagnostics 2001; **Las Vegas, NV**	Medical Graphics Corporation, Mari Orke, (800) 950-5597, ext. 444, www.medgraph.com

Notices

Scheduled Professor's Rounds 2001

Program #3 Noninvasive Ventilation: The Latest Word—Dean R Hess PhD RRT FAARC; Host Richard D Branson BA RRT FAARC—Video April 24 Audio May 29

Program #4 Patient Education for the Asthmatic—Tracey Mitchell RRT; Host Thomas J Kallstrom RRT FAARC—Video May 22 Audio June 19

Program #5 ARDS: The Disease and Its Management—Leonard D Hudson MD; Host David J Pierson MD FAARC—Video June 26 Audio July 17

Program #6 New Respiratory Drugs: What, When, and How—Joseph L Rau PhD RRT FAARC; Host Patrick J Dunne MEd RRT FAARC—Video August 14 Audio September 11

Program #7 Invasive Ventilation: The Latest Word—Richard H Kallet MS RRT; Host Richard D Branson BA RRT FAARC—Video September 25 Audio October 16

Program #8 Test Your Lungs-Know Your Numbers-Prevent Emphysema—Thomas L Petty MD FAARC; Host David J Pierson MD FAARC—Video October 23 Audio November 20

Helpful Web Sites

American Association for Respiratory Care
http://www.aarc.org
— Current job listings
— American Respiratory Care Foundation fellowships, grants, & awards
— Clinical Practice Guidelines

National Board for Respiratory Care
http://www.nbrc.org

RESPIRATORY CARE online
http://www.rcjournal.com
— Subject and Author Indexes
— Contact the editorial staff
— OPEN FORUM; submit your abstract online

Asthma Management Model System
http://www.nhlbi.nih.gov

Keys to Professional Excellence
http://www.aarc.org/keys/

Committee on Accreditation for Respiratory Care
http://www.coarc.com

The National Board for Respiratory Care— Examination Fees for 2001

Examination	Examination Fees
CRT	$190 (new applicant) $150 (reapplicant)
Perinatal/Pediatric	$250 (new applicant) $220 (reapplicant)
CPFT	$200 (new applicant) $170 (reapplicant)
RPFT	$250 (new applicant) $220 (reapplicant)
RRT (Written & CSE)	$190 (new) $150 (reapplicant) written only $200 (new and reapplicant) CSE only $390 (new) $350 (reapplicant) both

For information about other services or fees, write to the National Board for Respiratory Care, 8310 Nieman Road, Lenexa KS 66214, or call (913) 599-4200, FAX (913) 541-0156, or e-mail: nbrc-info@nbrc.org

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Company	Product	Circle #	Phone	Page #
Bedfont Scientific	Smoking Cessation Programs	161	609-654-5561	435
DHD Healthcare	Vibratory PEP Therapy	102	800-847-8000	C4
Hans Rudolph Inc	Breathing Lung Model	131	800-456-6695	435
Masimo	Pulse Oximetry	137	800-243-3444	426
Methapharm Inc	Drug	114	800-287-7686	433
Novametrix	MARS™ Pulse Oximetry	140	800-243-3444	C2
Praxair Inc	Portable Oxygen	122	800-299-7977	428
Siemens Medical Systems	INFINITY Patient Monitoring System	129	800-333-8646	C3

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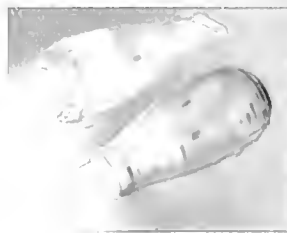
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